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GOITRE STUDIES: I. THE INCIDENCE OF ENDEMIC GOITRE IN THREE AREAS IN AUSTRALIA.

By F. W. CLEMENTS,
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THE study of the incidence and geographical distribution of endemic goitre in Australia is not new. The incidence in a number of localities has been previously reported,[1][2][3][4][5][6][7] whilst Harvey Sutton has summarized the geographical distribution of the condition in Australia.[8][9][10]

The present clinical survey had a dual purpose. The investigation in Gippsland was part of a comprehensive inquiry, in which an endeavour was made to elucidate some additional factors in the ætiology of endemic goitre; in this part of the work attention was especially directed towards the discovery of those conditions which resulted in the sporadic appearance of goitre even in a highly goitrous area. The extension of the survey to Canberra and Sydney was stimulated by a desire to know whether Canberra was an endemic goitrous area. The incidence of endemic goitre in Gippsland has been shown by a number of previous investigators to be high.[1][2][3][4][5][6] The incidence was known to be low in Sydney; what was the position in Canberra?

The Incidence in Gippsland.

Gippsland has been settled by a farming population for nearly one hundred years. During the past twenty-five years several studies have been made by medical officers of the Education Department, Victoria, into the incidence of goitre in the State,[1] and particular attention was paid to Gippsland. The present survey was not planned as a comprehensive investigation of the incidence of endemic goitre throughout Gippsland or even of a circumscribed locality, but was limited to children attending schools in a number of towns and hamlets that were particularly suitable for other aspects of this study (see Parts II, III and IV).[1]

The children examined were drawn largely from the farming community. For this reason the figures for the incidence of endemic goitre should not be taken as representative of Gippsland as a whole.

Procedure.

All children attending school and a number of pre-school children in the localities studied were examined; but the results have been given for those aged six to fourteen years, inclusive. Each child was given a general medical examination, during which particular attention was paid to signs of deficiency diseases as revealed by changes in the surface of the tongue, in the mucous membrane of the mouth, in the gums and lips, in the conjunctivæ and in the skin. The child's height and weight were recorded.

The degree of enlargement of the thyreoid gland was measured by palpation and by inspection from the front and side. The size of the gland was estimated and classified into one of the four categories recognized by Stocks:[11] 0, thyreoid impalpable; 1, thyreoid palpable, but not visibly enlarged; 2, thyreoid visibly enlarged, detectable from both the anterior and the lateral aspects, the enlargement not being sufficient to produce gross deformity of the neck; 3, thyreoid sufficiently enlarged to be considered "goitre" even by a lay observer.

The Results of the Survey.

The incidence of enlargement of the thyreoid in boys and girls classified into age groups is given in Tables I and II.

It will be noted that the maximum incidence in boys is in the nine to eleven years' age group, whilst in girls

[1] To be published later.

it occurs in the twelve to fourteen years' group. In general these figures are similar to those obtained in other surveys made in the Gippsland area.

The Incidence of Goitre in Canberra.

No previous surveys have been made of the incidence of goitre in Canberra. One explanation of this is the comparatively recent development of that city, for constructional work on essential services was commenced in Canberra in 1912 and the erection of homes and public

TABLE I.
Incidence of Goitre in Gippsland: Boys.

Age Group. (Years.)	Total Number in Group.	Thyreoid Palpably Enlarged.		Thyreoid Visibly Enlarged.	
		Number.	Percentage.	Number.	Percentage.
6 to 8 ..	172	40	23·2	23	13·4
9 to 11 ..	120	35	29·2	21	17·5
12 to 14 ..	61	17	27·9	8	13·1

TABLE II.
Incidence of Goitre in Gippsland: Girls.

Age Group. (Years.)	Total Number in Group.	Thyreoid Palpably Enlarged.		Thyreoid Visibly Enlarged.	
		Number.	Percentage.	Number.	Percentage.
6 to 8 ..	147	45	30·6	25	17·0
9 to 11 ..	119	34	28·6	30	25·2
12 to 14 ..	72	17	23·6	30	41·7

buildings was commenced in 1922, whilst Parliament and a number of Government departments were not transferred from Melbourne to Canberra until 1927. A considerable number of people were transferred there at about this time. The growth of Canberra in the intervening years is shown by the population figures given in Table III.

TABLE III.

Year.	Population.
1921[1]	1,150
1923	2,000
1925	3,500
1927	6,150
1929	6,880
1931	7,050
1933	7,330
1935	7,700
1937	8,410
1939	10,420
1941	12,200
1943	12,200
1945	12,800

[1] No satisfactory estimate prior to 1921.

Whilst a high percentage of the population is static, a significant portion remains in Canberra but a short time; this is due mainly to the nature of employment. Many of the male employees are public servants who are subject to transfer to other centres in Australia and abroad. This has meant that a proportion of the children at school in any one year are recent arrivals; however, with the passage of time there are more and more children in the upper grades at school who were born in Canberra. The routine annual medical inspections made in recent years by the schools' medical officers have revealed a number of children to have an enlarged thyreoid. These observations, together with the fact that Canberra is situated in the southern tablelands, which have been a suspected endemic goitrous area for many years, indicated that a goitre survey should be undertaken amongst the children living there.

Procedure.

The procedure followed was similar to that used in Gippsland. The standard by which enlargement was judged was the same and the study was undertaken by the same observer. In order to exclude recent arrivals, who would not have had the effect of local conditions, the survey was limited to children who had lived in Canberra for at least the previous two years.

The Results of the Survey.

The incidence of goitre in children in Canberra is shown in Tables IV and V. It will be noted that the incidence was found to be higher in girls than in boys, particularly in the twelve to fourteen years' age group.

TABLE IV.
Incidence of Goitre in Canberra: Boys.

Age Group. (Years.)	Total Number in Group.	Thyreoid Palpably Enlarged.		Thyreoid Visibly Enlarged.	
		Number.	Percentage.	Number.	Percentage.
6 to 8 ..	177	22	12·4	8	4·5
9 to 11 ..	198	49	24·7	11	5·6
12 to 14 ..	177	50	28·2	9	5·1

TABLE V.
Incidence of Goitre in Canberra: Girls.

Age Group. (Years.)	Total Number in Group.	Thyreoid Palpably Enlarged.		Thyreoid Visibly Enlarged.	
		Number.	Percentage.	Number.	Percentage.
6 to 8 ..	186	41	22·0	10	5·4
9 to 11 ..	160	59	36·9	16	10·0
12 to 14 ..	164	61	32·0	37	19·5

The survey in Canberra included children attending two secondary schools—namely, the Canberra High School and the Telopea Park Central School. The former provides education up to the Leaving Certificate examination and in general entrance is limited to children with a reasonable chance of benefiting from an education leading to professional and clerical positions. At the time of the survey the Telopea Park Central School provided education for those whose ability suggested a career limited to manual trades and similar callings. The standard of ability, personal hygiene and social development was, in general, higher in the high school than in the central school.

During the survey the impression was gained that enlargement of the thyreoid occurred more frequently and was greater in the girls attending Telopea Park Central School than in those at the Canberra High School. The relevant figures are given in Table VI.

The data in Table VI were subjected to statistical treatment by the χ^2 test, and this showed that the higher incidence of visible enlargement at Telopea Park Central School was significantly different from the incidence at the Canberra High School and was not due to chance sampling. At present it is not possible to state the cause of this difference.

The Incidence of Goitre in Bondi (Sydney).

During the surveys in Gippsland and Canberra it was thought that a need existed to examine a group of children living in an area reputed to be free of goitre. In collaboration with Dr. A. E. Machin, Principal Medical Officer

of the Department of Public Instruction of New South Wales, two schools in Sydney were selected. The choice fell on Bondi School and the Domestic Science School, Dover Heights. A high percentage of the children attending both schools reside in the suburbs along the coast, south of Port Jackson. The procedure and classification of thyreoid enlargement was the same as for Gippsland and Canberra. During the course of the actual survey it was found that a number of the children

TABLE VI.

Comparison of Incidence of Goitre in Girls Aged Twelve to Fourteen Years in Two Secondary Schools in Canberra.

School.	Number of Girls Examined.	Thyreoid Enlargement.		
		No Enlargement.	Palpable Enlargement.	Visible Enlargement.
Telopea Park Central School ..	37	13	13 (35%)	11 (30%)[1]
Canberra High School	69	39	23 (33%)	7 (10%)[1]
	106	52	36 (34%)	18 (17%)

[1] Differences statistically significant.

attending these schools had been born and had lived for some years in areas of Australia known to be goitrous. In the presentation of the results these children have been separated from those born in Sydney.

The Results of the Survey.

The incidence of goitre amongst the children examined at these two schools is given in Tables VII and VIII. The number of cases and the percentages in Tables VII and VIII indicate the number of children with goitre;

TABLE VII.

Incidence of Goitre in Bondi (Sydney): Boys.

Age Group. (Years.)	Total Number in Group.	Thyreoid Palpably Enlarged.		Thyreoid Visibly Enlarged.	
		Number.	Percentage.	Number.	Percentage.
6 to 8 ..	176	8 (0)	4·5 (0)	0	0
9 to 11 ..	155	2 (0)	7·7 (0)	2 (0)	1·3 (0)
12 to 14 ..	236	25 (13)	10·5 (5·5)	8 (0)	3·4 (0)

TABLE VIII.

Incidence of Goitre in Bondi (Sydney): Girls.

Age Group. (Years.)	Total Number in Group.	Thyreoid Palpably Enlarged.		Thyreoid Visibly Enlarged.	
		Number.	Percentage.	Number.	Percentage.
6 to 8 ..	136	9 (6)	6·5 (4·2)	2 (2)	1·4
9 to 11 ..	154	16 (8)	10·3 (5·0)	9 (5)	5·8 (3·3)
12 to 14 ..	293	40 (14)	13·6 (8·9)	25 (17)	8·5 (5·8)

the figures in parentheses represent the children who had been born in Bondi or the surrounding districts and had lived there throughout their lives. The differences between the figures inside and outside the parentheses represent children with goitre who had been born in other places, many of them known to be endemic goitre areas, and who had changed their residence to the Bondi district. In all cases the transfer had taken place within the last three to five years—that is, at such an age that the child could have developed the goitre in the area in which he or she had been born.

The separation of the figures revealed that a small percentage of both boys and girls who had been born in seaside suburbs and had lived there all their lives had enlargement of the thyreoid; in some cases the enlargement was visible. The enlargement of the thyreoid in the remainder had apparently occurred through residence in other localities.

The Significance of Enlargement of the Thyreoid in Adolescent Girls.

Before we proceed to compare the incidence of goitre in the three areas surveyed, it is necessary to inquire into the significance of thyreoid enlargement in adolescent girls at about the time of the menarche. Not a few practitioners when asked by an anxious parent to comment upon visible enlargement of the thyreoid in a girl twelve to sixteen years of age remark that it is a "normal" feature of growth at that age.

This problem has been the subject of at least two inquiries. Stocks[11] found that where goitre is not endemic the thyreoid gland changes very slightly in size from ten and a half to thirteen and a half years of age in boys, whereas in girls a rapid development of the gland occurs between thirteen and a half and fifteen years. This enlargement may not become noticeable. Ryle[12] investigated the incidence of goitre in school children, aged eleven to fifteen years, in four localities in England. His figures are reproduced in Table IX.

TABLE IX.

Locality.	Mean Percentage Incidence of *Visible Glands.*
Devon	26·0
Oxfordshire	17·5
Windsor	6·5
Essex	2·5

These differences are statistically significant.

It would seem that in girls during early adolescence the thyreoid gland is called upon to increase its output as part of the increased endocrine activity of that period. If the iodine reserves of the body and the iodine intake over these years are adequate to meet the increased demand, only a slight hyperplasia of the thyreoid occurs, and this passes unnoticed by either the general practitioner or the medical officer making a special goitre survey. However, if the reserves are low, owing to long residence in a goitre area, and if the intake is below optimal requirements, the thyreoid undergoes appreciable enlargement to trap additional quantities of iodine, as in Devon and Oxfordshire. These findings suggest that practitioners should hesitate to attribute an enlarged thyreoid in an adolescent girl to the menarche; it is much more likely to be due to iodine deficiency and thus be a manifestation of endemic goitre.

The ultimate size of the gland will depend upon at least two factors: (i) the actual demand by the whole body for thyroxine as part of the growth pattern of the individual, and (ii) the supply of iodine.

Whatever these factors are, the following conclusion reached by Ryle is pertinent to this inquiry:

> The percentage incidence of visible glands in an adolescent population is a useful index of its iodine consumption and of the goitre hazard to which it is exposed in later life, whether we regard the clinical findings as an early sign of specific deficiency—i.e. as pathological—or as expressing the more extreme limits of physiological variation or adaption.

Comparison of the Incidence of Goitre in Gippsland, Canberra and Sydney.

In this study Canberra has been treated as the unknown, Gippsland as the goitre area, and Sydney as the non-goitre area. The comparisons were studied statistically by the χ^2 test, and the results of the probability are given in Table X, with an indication as to whether a difference

TABLE X.

Comparison of Incidence of Goitre in Gippsland, Canberra and Bondi; Probability that the Difference is Significant.

Places Compared.	Boys. (Years.)			Girls. (Years.)		
	6 to 8.	9 to 11.	12 to 14.	6 to 8.	9 to 11.	12 to 14.
Sydney-Canberra ..	$p^1<0{\cdot}001$ Significant.	$p<0{\cdot}001$ Significant.	$p<0{\cdot}001$ Significant.	$p<0{\cdot}001$ Significant.	$p<0{\cdot}001$ Significant.	$p<0{\cdot}001$ Significant.
Canberra-Gippsland	$p<0{\cdot}001$ Significant.	$p<0{\cdot}001$ Significant.	$p<0{\cdot}01$ $>0{\cdot}05$ Significant.	$p<0{\cdot}001$ Significant.	$p<0{\cdot}3$ $>0{\cdot}2$ Not significant.	$p<0{\cdot}1$ $>0{\cdot}05$ Not significant.

[1] Probability.

is statistically significant. These data show that the incidence of goitre in Canberra is significantly greater than the incidence in Sydney for all age groups of both sexes. When compared with that in Gippsland, the incidence is significantly less amongst boys six to eleven years old and amongst girls six to eight years old; but there is no difference in the incidence amongst boys twelve to fourteen years old and girls nine to eleven and twelve to fourteen years old. In other words, amongst children in the last three groups the incidence of goitre in Canberra is as great as it is in Gippsland. From these figures Canberra must be considered an endemic goitrous area.

Summary.

1. A survey was made of the incidence of endemic goitre in school children in Gippsland (a known goitre area), in Sydney suburbs (a presumed non-goitre area) and in Canberra (the unknown) by one investigator in the spring of 1946. All three areas were visited over a period of six weeks.

2. The survey confirmed the premises with respect to Gippsland and Sydney, and showed that in Canberra the incidence of goitre was significantly greater than the incidence in Sydney for all age groups of both sexes. When compared with that in Gippsland, the incidence is significantly less amongst boys six to eleven years old and girls six to eight years old; but the difference in the incidence in the other age groups was not significant. That is, in the later age groups the incidence of goitre in Canberra is such as to indicate that the city is built in an endemic goitre area.

3. A significant difference in the incidence of goitre in girls twelve to fourteen years of age attending the two secondary schools in Canberra was recorded, the incidence amongst those at Telopea Park Central School being higher than amongst those at the Canberra High School.

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(4) "Goitre in Victoria", Report of the Minister of Public Instruction, Victoria, for the Year 1915-1916.

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(9) H. Sutton: "Geographical Control of Disease in Australia", Report of Hobart Meeting, 1928, Australian Association for the Advancement of Science, 1929, page 480.

(10) H. Sutton: "Geographical Incidence of Disease in Australia", *The Australian Geographer*, Volume II, 1933, page 3.

(11) P. Stocks: "Goitre in Adolescence. An Anthropometric Study of the Relation between the Size of the Thyroid Gland and Physical and Mental Development", *Biometrika*, Volume XIX, 1927, page 292.

(12) J. A. Ryle: "The Meaning of Normals", *The Lancet*, Volume I, 1947, page 1.

SINUSITIS AND SHORT-WAVE THERAPY.

By WILKIE SMITH, M.B., B.Sc.,
Sydney.

THE problem of sinusitis is daily before the eyes of every medical man, no matter whether he is actively concerned in treating it or is just a passive onlooker, and I propose to present my conception of the disease and its treatment tonight.

The nasal accessory sinuses are 14 in number: two maxillary sinuses, two frontal sinuses, two sphenoid sinuses, four anterior ethmoid sinuses and four posterior ethmoid sinuses. Occasionally a third anterior and posterior ethmoidal cell is found. The maxillary sinuses are present at birth, the frontal sinuses appear at about the fourth or fifth year, and the sphenoid sinuses about the third year and, according to one observer, are pneumatized at the seventh year. The ethmoidal cells are considered to be pneumatized at birth. All sinuses are lined by ciliated epithelium, the cilia of which direct discharge and foreign matter to flow towards the ostium of the sinus. The maxillary, frontal and anterior ethmoid sinuses drain into the middle meatus, and the posterior ethmoid and sphenoid sinuses into the superior meatus. In an experiment of C. C. Hilding, ink was placed on the floor of the maxillary sinus, in which for observation purposes a window had been opened to the inferior meatus; it was found that none of the ink passed through the artificial window, but it all flowed to the normal opening or ostium of the sinus. It was considered that any drainage through the antrostomy opening would occur only as the result of gravity, which could take place with suppuration. Most observers agree that the ostium of the maxillary sinus is badly situated and does not provide for adequate drainage and ventilation. Its position may be all right for the ape; but on the assumption of the erect attitude the ostium was situated too high for effective drainage and too obstructed for effective ventilation. However, there is much to be said for the wisdom of the good Lord, who placed it away from the direct effects of air currents and consequently away from direct bacterial invasion and the traumatic effect of cold air. Further, in respect to drainage it must be remembered that man spends about one-third of his life in the supine or prone position, and consequently drainage is assisted by sleep.

Causes of Sinus Infection.

Oto-laryngologists believe that if the sinuses were open more to ventilation and drainage, inflammatory processes within them would occur less frequently, and they conclude that the first principle in treatment of infection is to establish ventilation and drainage.

The causes of sinusitis may be either (*a*) endogenous or (*b*) exogenous.

Endogenous Causes.

Endogenous causes include the following: (i) All constitutional diseases, such as syphilis and diabetes, and diseases due to faulty metabolism. (ii) Deficiency diseases,

[1] Read at a meeting of the Australian Association of Physical Medicine on August 24, 1947, at Sydney.

ILLUSTRATIONS TO THE ARTICLE BY DR. WILKIE SMITH.

FIGURE I.—Case I: Both antra and right frontal sinus dull.

FIGURE II.—Case I: One month after treatment. Air appearing in both antra.

FIGURE III.—Case I: Four months after treatment. All sinuses clear.

FIGURE IV.—Case II: Mucosal thickening and dulness in all sinuses.

FIGURE V.—Case II: One month after treatment.

FIGURE VI.—Case II: Nine months after treatment.

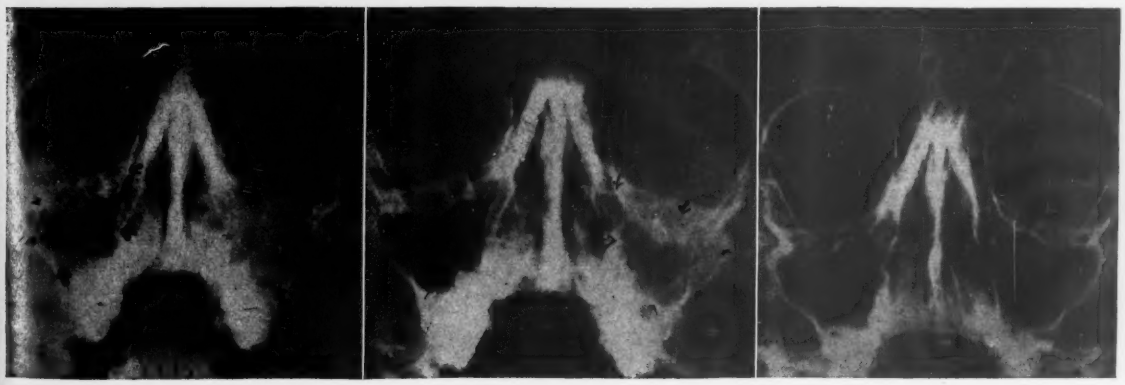

FIGURE VII.—Case III: Both frontal sinuses dull, left antrum dull, mucosal thickening right antrum.

FIGURE VIII.—Case III: Three weeks after treatment; right antrum clear, air in left antrum, frontal sinuses improving.

FIGURE IX.—Case III: Eighteen months after first treatment. All sinuses clear.

ILLUSTRATIONS TO THE ARTICLE BY DR. WILKIE SMITH.

FIGURE X.—Case IV: Mucosal thickening right antrum.

FIGURE XI.—Case V: Right frontal and right antral suppurative sinusitis.

FIGURE XII.—Case VI: Mucosal hyperplasia right antrum.

FIGURE XIII.—Case IV: Note increased mucosal thickening in reaction to treatment.

FIGURE XIV.—Case V: Result after two courses of treatment at intervals of six months.

FIGURE XV.—Case VI: Both antra clear after six weeks' treatment.

FIGURE XVI.—Case VII: Empyema in both antra; note fluid level.

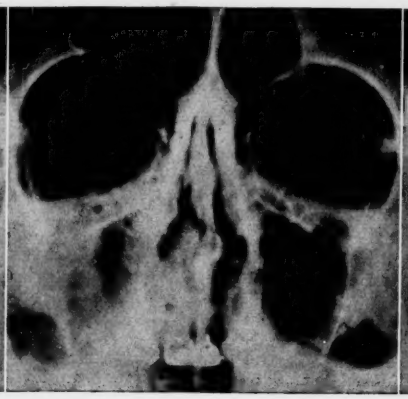

FIGURE XVII.—Case VII: After five weeks' treatment.

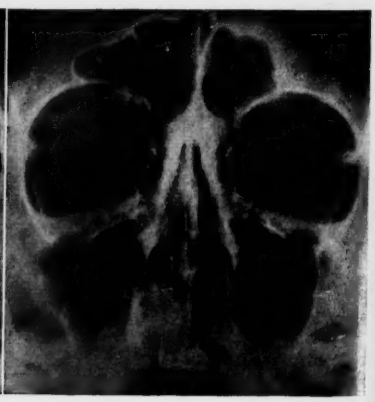

FIGURE XVIII.—Case VII: After six weeks' treatment; both antra clear.

such as hypovitaminosis and avitaminosis, and hormonal dystrophies with increased or diminished hormonal activity. (iii) Blood-borne allergens, the principal of which are fats or their metabolic derivatives, especially the fat of cow's milk and olive oil. Proteins, usually considered active allergens, appear to have little bearing on sinus infection; but further investigation is required in this respect. (iv) Excessive vasomotor stimulation, caused by fear, anger, anxiety and abnormal sex activity. Apparently the sympathetic nervous system is stimulated and vasoconstriction of the naso-sinusal cell results, followed by dilatation, which leaves the sinus cell vulnerable to bacterial invasion. (v) Contiguous infection or lymphatic drainage from a septic upper bicuspid or first molar tooth, which may lead to infection of the maxillary sinus.

Exogenous Causes.

Exogenous causes include the following: (i) Trauma by accident or direct surgical interference to the sinusal mucosa or underlying periosteum. This surgical misadventure is the commonest cause of all cases of refractory sinusitis. (ii) The presence of foreign bodies in the nasal cavities, such as nasal dressings. (iii) The instillation of nasal drops, usually vasoconstrictors, which, contaminated after touching the external nares and vestibule, may carry infection into the sinuses. Prolonged use of vasoconstrictors may exert a traumatic influence on the naso-sinusal cell and produce static inactivity of the cell and its cilia, followed by vasodilatation. (iv) Trauma from extraction of teeth and subsequent infection. (v) Swimming in contaminated water, whether fresh or still, as well as the inhalation of water infected by passage over the nares or vestibule. (vi) Climatic causes, such as persistent cold, wet weather and sudden changes of temperature. (vii) Exposure. (viii) Draughts. (ix) Lack of humidity in heated rooms. (x) Absence of sunlight and fresh air. (xi) Too much clothing or too little clothing. (xii) A damp scalp from wetting the hair, and insufficient covering to the head. (xiii) Living in damp dwellings.

Exciting Causes.

Exciting causes include the various microorganisms involved and those causing exanthematous and other infectious fevers. Hajek holds that the influenza virus is probably the most frequent source of infection. I believe that the virus is a common cause of sinusitis in epidemic form, but that most recurring sinusitis is due to the common organisms, especially the streptococcus. In 167 cases of sinusitis in which post-nasal discharge was examined, the following organisms were found: streptococcus, 120 cases; *Staphylococcus aureus*, *Staphylococcus albus* and *Staphylococcus citreus*, 30 cases; hæmolytic streptococci, six cases; *Streptococcus viridans*, eight cases; *Micrococcus catarrhalis*, 19 cases; pneumococci, 10 cases; diphtheroids, two cases; *Hæmophilus influenzæ*, two cases. Of over 3000 patients with sinus infection examined in the past seven years, of whom more than 1000 have been radiologically examined, about 40% show structural nasal deformities and about 50% no apparent deformity, and about 10% come under the heading of asthma, hay fever, eczema syndrome with or without deformity. In patients showing structural deformity there is a narrowing of the external nares, and partial closure of the nasal cavities is effected by buckling of the septum, hypertrophy of the turbinate mucosa and approximation of the lateral nasal wall to the twisted septum, which is Nature's attempt to present the least possible area of mucosa to the outside air. The hard palate is arched and its buccal surface is deeply concave, and the upper teeth are crowded, and often the first and second bicuspid and upper canine teeth are thrown out of alignment (see Figures XIX and XX). The same dental deformities may be found in the lower jaw. In later life the bicuspids are usually the first to show caries. I believe that these cases are those referred to by A. Thornton Taylor as the "retardation" type. One opinion is that this deformity is due to the fact that the nasal bones fail to develop *pari passu* with the hard palate and the structures forming the dental arch, and this disharmony of growth is considered responsible for causing the septum to buckle. However, I believe a likely cause is primary deformity of the nasal structures arising from the effect of endogenous allergens (commonly cow's milk) on the nasal mucosa, and exogenous trauma, such as the effect of cold air, on the same tissue. The mucosa endeavours to protect itself from the onslaught of cold air as a traumatic agent, and in so doing presents the least possible surface to the outside world. The septum in adapting itself to this bilateral trauma buckles and pulls in an upward direction the hard palate, and in so doing deepens the concavity of the hard palate and narrows the buccal arch. The teeth irregularities follow, with disuse as a contributory cause (see Case IV, Figures X, XI, XIX and XX). So in these cases there appears a definite relationship between cow's milk and the deformity, and the

FIGURE XIX.

FIGURE XX.

fat of the cow's milk or its metabolic derivatives is the responsible allergen. I am informed that pigs fed on cow's milk or skimmed milk (0·5% fat) develop the usual symptoms associated with the common cold—coryza, sneezing and cough—and that some pig farmers are disinclined to feed their pigs on this food even as a gift. Todd and Stock prohibit cow's milk to the asthmatic, and this information led me to draw a parallel between the asthmatic and the sinus sufferer, knowing that each was predisposed to the common head cold and that the following signs and symptoms were also common to both: low systolic and diastolic blood pressure, an anxiety state, a sensitive nervous system, dyspepsia, small nares and small nasal cavities. Attention was then centred on cow's milk, not only as having a relationship to sinusitis, but also as to the general use made of it in the average diet. From the important part it plays in the diet of the invalid and the infant, it has in this country become the food more publicized than any other as inseparable from good health. The medical profession has sponsored its claim to importance on its food value and its calcium content, and upon experimental investigation in which it was shown that school children given milk as an addition to their ordinary diet put on weight and appeared in better health than controls on the same diet without the addition of milk. It is quite probable that had the children been given a supplementary diet of balanced food without cow's milk, they would have progressed just as favourably, if not better, than those who received milk as a supplement. In England a "milksop" has been a derisive term for generations, and throughout most of its historic time England has been a beer-drinking and beef-eating country, and probably little milk was consumed until this century. Further, its use appears to encourage the temptation to depend upon soft food as supplying the needs of the body, with a liberal amount of sugar added, which goes hand in hand with milk as the basic breakfast food. As for the calcium content of milk and its importance in dental nutrition, reference to the uncivilized Australian aboriginal and to the islanders north of Australia will sufficiently answer the question whether good jaws, sound teeth and normal nasal structures can develop without milk. Structure and function run hand in hand; destroy one and the other will die. Take away the principal masticatory function of the jaws and teeth and it is not difficult to visualize a race of Australians with fewer teeth than exist

at present, with the bicuspids and the canines as the first to be lost. These are always the first to show signs of atrophy and of irregularity in alignment.

Of those cases in which no apparent nasal deformity is present, the cause of sinusitis is usually to be found in the conditions under which patients live and work. There is usually a history of working for long hours behind a shop, or in a kitchen or factory or home where there is inadequate ventilation or light. Apparently in these cases there is a relationship to atmospheric oxygen, carbon dioxide and humidity. Exposure to conditions in which humidity is regulated, as in central heating, is without doubt a frequent source of sinusitis.

In cases in which there appears to be a relation to the asthma, hay fever, prurigo syndrome, sinusitis is usually demonstrated radiologically at an early stage, and later on in this paper I intend to state that sinusitis is commonly the primary cause of asthma.

Other important causes of sinusitis of which mention must be made are the use of intranasal drops, which may act as a vehicle carrying infection to the sinuses. The region of the external nares is a contaminated area and any fluid passing over this region is likely to convey infection to cryptic cavities. Water entering the nostrils through swimming or immersing the head in water comes under this category and is a common source of sinus infection. Douching of the nose likewise is a cause, as well as the use of most proprietary preparations dispensed as nasal drops. Most of these nasal drops are vasoconstrictors, which, if used injudiciously, may act as traumatic agents and may also convey infection from the external nares and the vestibule. In respect to cases coming under the heading of undue sex activity, it is wise when one is at a loss to locate a cause to think of the French: *"Cherchez la femme."* Over-indulgence in intercourse, in my opinion, is often a contributory cause of sinusitis. The chain of events producing this is possibly over-stimulation of the pituitary gland with hormonal activation of the sex glands, thence stimulation of the adrenals and the sympathetic nervous system, followed by vasoconstriction of the naso-sinusal mucosa and later vasodilatation. The mucosa is weakened and the way is prepared for bacterial invasion.

Proof-puncture of the antra is not without danger, and surgical interference, whether simple or radical, produces the most difficult of all forms of sinusitis to treat.

Complications.

The commonest complications are acute and chronic tonsillitis, cervical adenitis, Eustachian catarrh, *otitis media*, adenoiditis, laryngitis, bronchitis, bronchiolitis, bronchiectasis and asthma, fibrositis and arthritis.

I believe that most forms of acute tonsillitis are secondary to sinusitis, and that the tonsil is the first barrier in defence of the primarily infected sinus. In other words, an inflamed tonsil is usually a beacon light indicating that sinus infection is present. This opinion is based on two facts: (i) when lipiodol is placed in the maxillary sinus it is later found in the tonsil and passes by lymphatic drainage to the tonsil; (ii) in most cases acute tonsillitis will clear within two days after treatment of the nasal accessory sinuses by short-wave therapy.

One may also hazard the opinion that septic discharge from a sinus passing over the tonsil may infect it. The editors of "The 1946 Year Book of the Ear, Nose and Throat" have a different opinion. They make the following statement:

> There is abundant evidence to suggest that upper respiratory infections begin in the lymphoid tissue of the naso-pharynx and that the excess nasal secretion and swelling of nasal mucosa are secondary.

In fairness to this opinion, which is based on the relief afforded from treatment of the lymphoid tissue of the naso-pharynx by surgery, deep X rays and radium tamponage, it could be argued that this tissue was favourably influenced by the use of short-wave therapy when the patient came into the condenser circuit. Possibly short-wave therapy acts in much the same way as radium. However, the correlative radiological evidence of sinusitis in these cases is very convincing, and also the fact that when the tonsils are removed attacks of sinusitis are more severe and more apparent, and respiratory infections are much more common.

That the respiratory lesions are secondary to sinusitis may be concluded from the fact that they clear up when the sinus infection is successfully treated with short-wave therapy.

Asthma is considered a complication of sinus infection following control of this condition by the application of short-wave therapy to the nasal accessory sinuses. To understand asthma, one must examine the patient when bronchial spasm first becomes evident, whether this occurs at three years of age or is a sign apparent for the first time at forty years of age. Always there is a history of recurrent head colds with sore throats. About the third day after the onset of the head cold there develops an attack of asthma, to be followed in a few days by bronchitis. This is the early picture, which is altered considerably with increasing frequency of asthmatic paroxysms. The head cold becomes less apparent, the nose ceases to discharge, there is diminished resistance in the sinus mucosa, the lymphoid tissue of the naso-pharynx ceases to become inflamed and hypertrophy of the cervical lymphatic glands is not found. A chronic asthmatic never has acute or chronic tonsillitis, and in asthmatics it is rare to hear of appendicitis. This evidence supports the view that the resistance put up in the early stages of asthma by the nasal accessory sinuses, the tonsils and the lymphoid tissue of the neck has completely failed, and asthma develops without any apparent initial clinical signs.

In spite of claims for psychogenic ætiology, and of the dicta of those who favour allergy as a principal cause, I believe asthma begins in the nasal accessory sinuses and that its course is determined by failure of the antigenetic lymphoid tissue of the body. In "The 1945 Year Book of the Ear, Nose and Throat" editorial comment is as follows:

> Much remains to be learned about the function of lymphoid tissue, and additional knowledge of this tissue will markedly change our present oto-laryngologic treatment, especially the operative removal of tonsils and adenoids in children. Evidence is accumulating that the chief function of lymphoid tissue in the respiratory and digestive tracts is to form antigens.

It is likely that infection tracks down from the sinuses, meets with little or no resistance from the lymphoid tissue of the neck, and travelling extrabronchially reaches the bronchial mucosa by the back door. The bronchial musculature goes into spasm, either in resisting invasion or in response to the influence of histamine. To explain those cases in which a spasm develops at the smell of food or sight of a flower or other psychogenic stimulus, one can only regard these as arising from the influence of the central nervous system on well-worn synapses and reflex arcs. The same would apply to the action of internal and external allergens. Evidence for the nasal origin of most asthmatic attacks may be found in an article by J. P. P. Stock, entitled "Short-Wave Therapy in the Treatment of Asthma". It has been noted in my series of cases that dermatological lesions in cases coming under the asthma, hay fever, prurigo syndrome have often disappeared in two or three days after treatment of the nasal accessory sinuses with short-wave therapy alone.

Other important complications of sinus disease are optic neuritis, retrobulbar neuritis, osteomyelitis of the sinus wall, extradural abscess and brain abscess.

Symptoms and Signs.

One of the main symptoms is headache, referred to the supraorbital, temporal and occipital regions. Pain may be experienced over the eyes, behind the eyes and over the face. Obstructed breathing and snoring may be present. Snoring in children is commonly caused by blockage of the ostia leading to the sinuses, and results from allergic inflammation of the mucous membrane in these regions and not from enlarged tonsils and adenoids, as is usually supposed. The œdematous membrane over the ostia acts as a valve in permitting air to pass through small openings into the sinus cells. Often a child as young as two years

of age has had the tonsils and adenoids removed as obstructive when the real obstruction was blockage in the ostia of the nasal sinuses. Pus may be seen on rhinoscopic examination in the nasal cavities and on the posterior pharyngeal wall. Tenderness on pressure may be present under the supraorbital region. Epistaxis and disturbances of vision and giddiness may be present, as well as toxæmia, malaise and inability to concentrate. Olfactory, visual and auditory disturbances may occur. A flush over the skin in the middle third of the nose is considered to be due to ethmoidal infection, and capillary dilatation in the same region to be due to chronic ethmoiditis. The objective signs may be seen by transillumination and X-ray examination, but transillumination should be discarded because it is most unreliable. Good technique in the use of X rays gives most informative results and supersedes other diagnostic measures commonly used. The presence of polypi, cysts and mucosal thickening in the sinuses can be accepted as evidence of past or present sinus disease.

Proof puncture commonly practised by oto-laryngologists can be misleading in cases in which macroscopic amounts of pus do not appear in the irrigating fluid.

Treatment.

Surgery.

For years the surgeon has worked on the idea that the nasal cavities require adequate ventilation and drainage when disease is present; this in spite of the fact that the closure of these cavities appears purposeful on the part of Nature and designedly defensive. The two principal factors responsible for this closure in patients coming under my observation seem to be trauma from an endogenous allergic factor (commonly cow's milk in this country) and exogenous trauma in the form of cold air. To open pathways to the sinuses and make artificial openings into their cavities would appear invalid treatment if allergens continue to exert a traumatic influence on the naso-sinusal cell. Evidence of this may be seen when Nature proceeds to close these surgical openings a few months after they have been made.

However, in recent years the surgeon has become much more conservative, and whereas twenty years ago it was common practice to exenterate the ethmoid sinuses, make external openings into the frontal sinuses, remove one or more turbinates and indiscriminately perform simple and radical antrostomy, these procedures are infrequently carried out today. This is largely the result of the Hajek Clinic in Vienna, which found that patients who refused surgical treatment to their ethmoid and other sinuses had returned later to the clinic fully recovered from the condition for which operation had been advised. A further influence was the mortality rate arising from meningitis following surgical trauma, and generally unsatisfactory results. In discussing intranasal and external surgery of the sinuses, the editors of "The 1946 Year Book of the Ear, Nose and Throat" state that "unless the symptoms call for immediate action, it is usually wise to postpone radical operations until all simpler measures have been given a trial". With the use of modern apparatus conveying short-wave therapy it is now possible to say that surgery of the sinuses is unnecessary.

The most pressing symptom to be relieved in sinus infection is pain, and this is best achieved by correct technique in the use of short-wave therapy and not by surgery. Fear is sometimes expressed that the complications of osteomyelitis and brain abscess are likely to develop unless recourse is had to the knife; but even with this fear in mind, short-wave therapy in combination with chemotherapeutic agents is the treatment to be employed, and one need have no fear of being challenged on this point when one reviews the issue in a series of over 3000 cases. For a period I used proof puncture as collateral treatment with short-wave therapy and washed out the antra first with saline solution, then with sulphonamides, and later with penicillin in solution; but in no case was I convinced of any advantage gained over the use of short-wave therapy alone. In fact, as was stated before, often surgical trauma to the sinus wall leaves the patient in a condition refractory to the more simple forms of treatment. There is an endless stream of patients to be found who walk the streets with symptoms which would never have developed if recourse had not been had to surgical treatment of the sinuses.

Short-Wave Treatment.

In the translation of the New Testament made by Dr. Moffit appears the following:

> The slight trouble of the passing hour results in a solid glory past all comparison for those of us whose eyes are on the unseen, not the seen; for the seen is transient, the unseen eternal.

It may appear inappropriate to use a statement of this character to exalt the virtues of an unseen force used as a physical agent in treatment of diseased tissue, but such is the importance ascribed to short-wave therapy. In the past few years we have seen great unseen forces at work, harnessed in a way never dreamt of before, carrying benefits and destruction to a world uncertain of its end. Short-wave therapy is one of these forces, first used as a medium in the transmission of the human voice to all parts of the globe, but later used in a manner of which few medical men are sufficiently appreciative as a physical agent in treatment of disease and traumatic conditions. According to Stock:

> Short-wave in its application to the human body (1) inhibits the activity of bacteria, (2) stimulates the body's defensive processes, and (3) quickly causes shrinkage of a swollen mucosa, particularly if this swelling is allergic in origin.

Short-wave therapy by thermal activity stimulates ciliary movement, produces considerable hyperæmia, promotes drainage, disperses scar tissue and regenerates cells diseased by the common microorganisms. According to another author:

> Its biological reaction is dependent upon the heat, the wave-length, the specific conductivity and other specific factors of the substance; its dialectic constant, dissociation, the dimensions of the condenser field, of the energy supplied and many more. A solution of albumen coagulates under ordinary conditions of about 75 degrees Centigrade. In the condenser field coagulation takes place at a temperature as low as 60 degrees.

In modern usage, spaced electrodes are employed, producing in between a condenser field, so preventing direct passage of the current through the tissues. Before the war, Stock, in collaboration with Professor Burch, of Bristol University, developed a rotating electrode, believing that the condenser field was cylindrical. By rotating one electrode he thought more uniform heating of the tissues would take place. He later admitted errors in experiments which gave birth to the rotating electrode, but continued to perfect its use because of improved results in the treatment of sinus disease and asthma. Stock and Burch state that "the only way to heat the whole of the surface of a cavity is to change the direction of the lines of flow of the current periodically", and considered that "this is a reason why it may be an advantage to use a moving electrode in treating the cavities associated with the nose". Experiments were conducted with stationary electrodes and the rotating electrode on the same patient. First the stationary electrodes were used and heat was applied to the nose. The temperature before treatment in the posterior ethmoidal region was 98·2° F., and after ten minutes' treatment with heat applied to the limit of skin tolerance there was no rise in temperature. On the same patient he used the rotating electrode and applied heat for the same period of time. The temperature before treatment was 98·2° F. and after treatment 102·6° F., a rise of 4·6°. From this he concluded that the depth dose in respect to temperature rise was higher with the rotating electrode than with the stationary electrode.

In the treatment of chronic inflammatory conditions better results were obtained by the use of greater current strengths in the patient circuit than in acute conditions, and in order to give effect to this Stock had graduated electrodes made with varying capacities. These provided for the application of relatively weak doses of short waves

to begin with and enabled currents of greater strength to be used as treatment progressed. When the rotating electrode is used, it can be said that in acute sinusitis relief is obtained more quickly than by any other form of treatment, and treatment is helped by the use of the sulphonamide drugs and penicillin. In the treatment of subacute and chronic sinusitis the results are far better than when surgery and other forms of treatment are used. Most failures come from that large group of patients on whom some form of surgical treatment has been employed, and this includes irrigation following proof puncture, and those patients who are still continuously exposed to the precipitating cause. B. G. Wade, in a recent lecture on sinusitis, stated that before treatment was begun drainage of a sinus might be necessary as well as turbinectomy and resection of the nasal septum. This has not been my experience, since I now treat all infections of the sinuses without preliminary drainage, and I do not advocate turbinectomy because short-wave therapy shrinks the turbinate mucosa. As for resection of the nasal septum, this is not recommended, because (i) few surgeons remove that portion of the septum causing obstruction—that is, the upper portion—and (ii) the use of short-wave therapy shrinks the septal mucosa, and help is also afforded by eliminating causative factors such as endogenous allergens. In my opinion, surgical treatment of the sinuses should be relegated to that period of medical history before the use of short-wave therapy was thought of, and short-wave treatment should be persisted with even though in some cases progress may be slow.

Briefly, the following routine is adopted in the treatment of sinusitis. First an attempt is made to find the cause, and this is dealt with, although it may not be an easy matter. Cow's milk and fats are excluded from the diet empirically and for reasons already mentioned, even in cases in which allergy is not suspected. Doses of short-wave therapy employing the rotating electrode are given every second day for four to six weeks, when in most cases resolution will take place. Children as young as three years of age may be treated, and a course of three weeks is usually sufficient for them. Should the condition prove stubborn, a further course of six weeks' duration is given, electrodes of greater capacity being used. Progress X-ray pictures are taken at the end of six weeks, and further courses of treatment are decided upon after the progress as shown by X-ray findings has been reviewed. In what may be termed surgical failures, it is wise to give at least an annual course of short-wave therapy, as this will relieve most of the patient's symptoms. Vaccine therapy is always employed *pari passu* with short-wave therapy, as this helps to promote antibodies and resistance to the common cold. When acute pain is present, and in those cases commonly referred to as migraine (which is due in 90% of cases to sinus infection) it may be necessary to give short-wave treatment twice a day for two or three days to bring relief. Pain is one symptom for which one can dogmatically promise relief from short-wave therapy with the rotating electrode. In the treatment of Eustachian catarrh, acute middle ear disease, acute tonsillitis, acute laryngitis and respiratory infections, the application of short-wave therapy is directed first to the sinuses as the primary source of infection, and thence to the secondary complications. Many of these conditions clear up on treatment of the nasal factor alone. In asthma much the same plan is adopted, irrespective of whether lesions can be demonstrated radiologically in the sinuses. Intravenous "Pyrifer", a *Bacillus coli communis* suspension given intravenously, is being given a trial as a desensitizing agent.

When the lower floor of the antrum has been exposed through extraction of a tooth, no attempt should be made by the dental surgeon to interfere with the sinus mucosa and, should suppuration develop, the patient should forthwith be referred to a doctor practising physical medicine, who has the requisite equipment for treatment.

Surgical procedures on the tonsils and adenoids of children should never be carried out until the nasal accessory sinuses have been investigated, and it will be found that most infections of the tonsils will clear up with the application of short-wave therapy to the sinuses and with attention to allergic factors and to living conditions. If necessary, a septic tonsil can be placed in the condenser field, and healing will take place without recourse to surgery. Much the same can be said of the septic tonsil in adults. In a survey of 10,000 boys and 3500 girls during the period 1930-1934, the Medical Research Council of the Privy Council of Great Britain found that among the 56% of boys and 50% of girls who had had their tonsils removed, the sickness experience was no better than that of the children who had undergone no operation; in fact, there was some slight indication that the incidence of rheumatism, *otitis media* and mastoid disease was higher among the group whose tonsils had been removed. J. A. Anderson states that for a thirty-day period after tonsillectomy there is a predisposition to the development of bulbar or bulbo-spinal poliomyelitis, and that these two forms of the disease occurred sixteen times more frequently in the group subjected to operation than in the general child population. These facts should cause parents to give careful consideration before submitting children to the operative removal of tonsils and adenoids.

Since I have been treating the sinuses with short-wave therapy as the primary source of aural infections, acute mastoid inflammation has never been seen, and I have not performed myringotomy for acute middle ear disease for the past five years, being satisfied to treat this condition with the short-wave rotating electrode in combination with chemotherapeutic agents. Should I encounter an acute mastoid condition, short-wave therapy would still be used to wall off infection, and there would be less fear of infection spreading to the meninges than if surgery was used.

If children suffer from a persistent cough which can with confidence be related to infected sinuses, promise of a cure can be made if these children are referred at an early age for short-wave treatment. In most cases chronic bronchitis, bronchiolitis and bronchiectasis set in before the age of three years and this is when the children should be sent to the doctor specializing in physical medicine, before irreparable damage has been done to lung tissue.

It cannot be too strongly urged that in the treatment of sinus infections the use of short-wave therapy should be restricted to medical practitioners, who by their training are best equipped to undertake investigation into ætiology and pathology and are experienced clinicians. Much harm could be caused by committing a patient to the care of one untrained in the basic sciences of morphology, physiology, pathology and physics, and the mere application of electrodes to diseased tissue will be doomed to failure unless regard is paid to this fact.

Other Treatment.

1. The argyrol pack as collateral treatment is advocated by Stock and others. This I have not found necessary.

2. Nasal drops containing ephedrine are sometimes used as decongestants and for drainage. These in my opinion have no healing properties nor do what they are supposed to do—promote drainage; but actually they may be a cause of sinus infection. Recently I conducted a series of experiments on the shellfish commonly known as the mussel. On placing the bar of the gill of this fish in saline solution under the microscope one could quite easily see ciliary movements. When the slide was heated above the temperature of tap-water, ciliary movement became much more active, as though from an electrical stimulus. When the solution was cooled with ice, movements were slower until finally they stopped. Next, a drop of "Neo-Synephrine Hydrochloride" was added to the saline solution and there was a dramatic cessation of all ciliary movement, so that one could count each individual cilia. Further, the whole field was static, and no movement of discharge or foreign matter was seen to take place. However, further perfusion with saline solution restored the normal ciliary movement. It was concluded that if the cilia lining the naso-sinusal cells behaved like the cilia of this shellfish under the influence of ephedrine, then it

was fraudulent to state that drainage was improved by the use of ephedrine preparations, when actually all activity came to a standstill. One author states that in no part of the body is infected tissue treated by the application of vasoconstrictive agents. For these reasons the use of vasoconstrictors is condemned, as well as for the fact that they may act as a vehicle carrying sepsis from the external nares to the sinuses.

3. Recently in America the pressure chamber has been used in the treatment of sinus infections, and good results are reported. In this the patient is exposed to a reduced atmospheric pressure, which has as its object suction of discharge from the sinus by a difference of pressure within and without the sinus.

4. Infra-red and ultra-violet rays are said to be of value; but as their depth penetration is but a few millimetres, it is difficult to see how they can reach the seat of infection in sinuses in which air separates the anterior and posterior walls.

5. Nasal douching is condemned, because organisms may be carried from the external nares into the sinuses. Under this veto come sea bathing and the immersion of the head under water. Sea bathing and bathing in rivers and still pools are contributory to sinusitis and acute middle-ear disease.

6. Vitamins in large doses are said to be of value, and perhaps they are in cases of sinusitis due to vitamin deficiency.

7. The use of sulphonamides and penicillin is of unquestionable value in acute and subacute cases, especially as collateral treatment with short-wave therapy; but I have not in any case seen chronic sinusitis successfully treated with these drugs alone.

Reports of Cases.

The following are reports of some illustrative cases.

CASE I.—A male patient, aged five years, was first examined in 1944; he had a history of bronchitis and asthma since the age of three years. For six months he had been confined to bed with recurrent paroxysms of asthma. One grandfather had suffered from asthma. Skin tests for allergy had been carried out and injections of the offending allergens had been given without relief. His white cell differential blood count showed that 7% of the leucocytes were eosinophile cells. There was also a history of bed-wetting, grinding of the teeth and head colds. X-ray examination (Figure I) showed both antra to be dull (a stain was present over one antrum); the right frontal sinus was just developing and was dull. After one month's short-wave treatment air began to appear in both antra (Figure II), and four months after treatment all visible sinuses were clear (Figure III). He was taken off cow's milk, of which he had three pints daily, and subsequently gained in weight and attended school regularly. Vaccine therapy was given simultaneously with short-wave treatment. He had one mild attack of asthma one year after treatment.

CASE II.—The patient was a male, aged eight years. His history was obtained from his father, who is a medical practitioner. From 1941 to 1944 the patient had suffered from frequent head colds, bronchitis and *otitis media*. His tonsils and adenoids were removed in 1941, and in the same year one antrum was washed out, and a double myringotomy was performed. In 1942 he suffered from bronchopneumonia and frequent head colds. His adenoids were removed again, and one antrum was washed out. In 1944 he had the same antrum washed out again, and mucus was present in the irrigating fluid. He continued to suffer from head colds with nasal discharge. Skin tests revealed sensitivity to lucerne and hen feathers, but injections of these allergens and of bacterial suspensions failed to give relief. After short-wave treatment, vaccine therapy and a restriction of fats, the father reported at the end of 1944 that "the changes clinically and radiologically are most pleasing, and he is far better now than at any time during the last three years". Later the father reported that the lad had had an attack of bronchitis with asthma. In September, 1944, examination of his blood revealed 5% of eosinophile cells in a differential white cell count, and in May, 1946, a count showed 12% of eosinophile cells. There is a history of hay fever on both sides of the family.

This case illustrates the mistake of removing adenoids and tonsils where established sinus infection is present, and also the error in attempting to desensitize the patient with the relevant allergens while infection is extant in the nasal accessory sinuses.

CASE III.—A female patient, aged nine years, was first examined in May, 1944, when she complained of blockage in the nose and a cough which had been present for four years. She had been fed on cow's milk since birth, and it was customary to take it with every meal. Large quantities of cheese and ice-cream were consumed. Rhinoscopic examination disclosed engorged turbinates, and suppuration was present in the upper nasal cavity. Pathological examination of post-nasal material revealed the presence of hæmolytic streptococci. The patient received short-wave therapy with the rotating electrode on alternate days for one month, and a restriction was placed on fats. Sulphathiazole tablets were prescribed in the early stage of treatment, and an autogenous vaccine of hæmolytic streptococci was given. The patient was free of symptoms after the first course of treatment. X-ray examination (Figure VII) shows dulness of both frontal sinuses and one antrum, and mucosal thickening of the right antrum.

CASE IV.—A female patient, aged fourteen years, was first examined in June, 1945, when she complained of blockage in the head, drainage down the back of the nose and affection of her speech. She had twinges of rheumatism in both knee joints and down her legs. As a baby she had been fed on a proprietary preparation, but never could take the full amount. She had always been a restless child and had trouble with every tooth. Her mother suffers from headaches and a blocked nose. There is a family history of asthma and tuberculosis on the father's side. At nine years of age the patient had vague abdominal pains, and her appendix was removed without relief of pain. At ten years of age her adenoids and tonsils were removed, and at this time she spent fifteen weeks in hospital with rheumatic fever. She had pains in the legs, ankles and elbow joints, and could not hold a glass of water. At twelve years of age proof puncture of both antra was performed. At fourteen years she spent six weeks in bed with bronchitis. A cough has been present since the age of nine years. After one month's short-wave treatment to the nasal accessory sinuses, a strict veto on cow's milk and fats, and vaccine therapy, she does not complain of a cough and pain as formerly. Figures X and XI, as also Figures XIX and XX, show the dental deformities referred to in the text.

This is sinusitis in its worst form—active, non-suppurative and not easily demonstrated radiologically or by proof puncture. Its activity is disclosed by its effect upon respiratory tissue, muscles and joints. It is this form of sinusitis which is most likely responsible for rheumatic fever and rheumatological conditions in general. The streptococcus is probably the invading organism, and milk allergy and cold are related ætiological factors.

CASE V.—A male patient, aged fifty-six years, was first examined in September, 1944, when he complained of intense frontal headaches and of a cough which had been present for over one year. He was a school teacher by occupation and supplemented his earnings by playing poker each night in a confined, smoky and airless room in the city. A first course of short-wave therapy with the rotating electrode of low dosage was given on alternate days for six weeks, and a second course six months later. His headache was relieved before the end of the first week's treatment, and his cough before the end of six weeks. X-ray examination of his nasal accessory sinuses shows the right antrum and the right frontal sinus to be dull (Figures XII and XIII).

CASE VI.—A male patient, aged fifty-four years, a priest by profession, gave a history of recurrent head colds for six years, and of laryngitis with loss of voice every winter. He complained of headaches and a blocked nose, and of overwork. He was very much overweight. X-ray examination of the nasal accessory sinuses revealed mucosal hyperplasia of the right antrum, which cleared up after six weeks' short-wave therapy (Figures XIV and XV).

It is interesting to note that this patient subsequently developed a severe head cold which short-wave therapy failed to relieve. On this occasion the organism recovered from post-nasal material was the hæmolytic staphylococcus. Previously he had received two applications of short-wave therapy every winter with relief of symptoms.

CASE VII.—A male patient, aged fifty-six years, a retired Indian Army officer, was first examined in June, 1947, when he complained of utter exhaustion. A cough had been present for at least three months. On examination of the patient no abnormal pulmonary signs were found, but pus was observed on the posterior pharyngeal wall. An X-ray

film of his nasal accessory sinuses revealed dulness of both maxillary sinuses, and the so-called fluid level was clearly defined. Proof puncture was not performed, nor were antral irrigations given. Treatment consisted of daily applications of short-wave therapy with the rotating electrode, at first with electrodes of low capacity with increasing dosage as treatment progressed. Complete recovery had taken place at the end of six weeks' treatment. X-ray examination showed the disappearance of pus from both antra. Sulphonamide therapy was also instituted, and bacterial suspensions were given on alternate days (Figures XVI, XVII and XVIII).

Conclusion.

In conclusion I can only say that little over a decade ago diathermy was hailed as an adjuvant of quackery and charlatanism; but the realization of its true therapeutic worth can best be shown by the fact that we are meeting here to night as a group of specialists, all of whom use this healing agency, and although we still must regard the problem of sinusitis and its treatment as seen through a glass darkly (to quote again from the Scriptures), yet I consider that modern short-wave therapy, with due attention to ætiological factors and appropriate chemotherapy when indicated, offers the sufferer much quicker, much less painful and certainly much more permanent relief than any surgical methods yet devised.

Acknowledgement.

In the preparation of this paper reference was made, amongst other authors, to Ballenger, writing in "Diseases of the Ear, Nose and Throat", and to notes taken from the teaching staff of the Hajek Ear, Nose and Throat Clinic of Vienna, where an internship was spent in 1933. Whilst acknowledging these and other sources of information, I desire to accept full responsibility for the opinions expressed in the paper generally.

INVESTIGATION OF CARBOHYDRATE METABOLISM IN NORMAL PREGNANCY.

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THE effect of pregnancy on the various metabolic processes of the human body has been the subject of vigorous research for a number of years. As far back as 1895 von Jaksch and Lanz showed that pregnant women developed glycosuria after the ingestion of glucose. In 1920 Frank and Nothmann held that the occurrence of glycosuria after glucose ingestion could be used as a test for the diagnosis of pregnancy. The reliability of the method was affirmed by Welz and Van Nest[1] and by Hirst and Long.[2] However, other workers obtained very different results. Schirokauer[3] found the incidence of alimentary glycosuria after a dose of 100 grammes of glucose to be only 27·6%. Hellmuth[4] and Scheffey[5] both decided that it was impracticable to administer large doses of glucose orally, and also that the method was unreliable as a test for pregnancy.

Many pregnant patients have been referred to this laboratory for investigation of glycosuria. While *diabetes mellitus* was found to be rare, the impression was gained that many of the glucose tolerance curves seemed to be abnormally low. Since the publications of previous workers did not give a complete picture of carbohydrate metabolism in pregnancy, it was decided to investigate the effects of the ingestion of glucose on a series of women in early and late pregnancy and some months after the pregnancy was over. The investigation was carried out under the following headings: (i) incidence of glycosuria in pregnant women under their normal conditions of diet; (ii) occurrence of glycosuria after a large dose of monosaccharide; (iii) fasting blood sugar level in pregnant women under their normal conditions of diet; (iv) the glucose tolerance curve; (v) the Exton Rose test.

THE INCIDENCE OF GLYCOSURIA IN PREGNANT WOMEN UNDER THEIR NORMAL CONDITIONS OF DIET.

I have been unable to find any statistics dealing with the incidence of glycosuria in the normal population or in women, but Blotner and co-workers[6] found that glycosuria occurred in 0·8% and diabetes in 0·5% in a large group of selectees and volunteers for the United States Army in the 1939-1945 war. Spellberg and Leff[7] in a similar group obtained the much lower values of 0·1% for glycosuria and 0·03% for diabetes. In a series of tests on normal non-pregnant women in this laboratory the urine of an exceptionally high percentage (39·5) produced a slight reduction of Benedict's solution, whilst that of only 6·9% produced reduction of Fehling's solution. The question then arose as to what amount of glucose present in urine would cause reduction of both of these solutions.

Benedict[8][9] found that Fehling's solution would indicate the presence of glucose only when it was in excess of 0·1%.

In this investigation it was discovered that a solution of glucose in water gave an entirely different type of reduction from that obtained when glucose was dissolved in urine.

With the use of serial dilutions of 1% pure glucose in urine which had been proved to be free of any reducing material, it was found that a slight reduction of Benedict's solution occurred when as little as 0·06% glucose was present, but that no reduction could be obtained with 0·03% glucose in urine. A concentration of 0·12% glucose was necessary to obtain even a slight reduction of Fehling's solution (Table I).

TABLE I.

Concentration of Glucose in Urine.	Reduction of Fehling's Solution.	Reduction of Benedict's Solution.
1·0%	+++	+++
0·50%	++	++
0·25%	+	++
0·12%	−	+
0·08%	−	+
0·06%	−	+
0·03%	−	−

However, it must be remembered that small amounts of reducing sugar are present even in normal urine, Moritz[10] stating the amount to be 0·05% and Harrison[11] 0·10%. This would account for the high percentage of positive reactions obtained with Benedict's test in the present series of urine specimens.

Therefore, since the glucose content of normal urine often lies above the threshold of sensitivity of the Benedict test, it seems preferable to apply the term "glycosuria" to the condition of patients whose urine contains glucose in sufficient quantity to reduce Fehling's solution. Although the strong alkali present in Fehling's solution may liberate ammonia from urea and also destroy small amounts of glucose, it is believed that the number of cases of glycosuria missed by this reaction would be smaller than the number of cases falsely assessed as glycosuria by means of a slightly positive Benedict reaction. The Benedict test is useful in confirming the reduction obtained with Fehling's solution and in eliminating the possibility that the reduction is due to uric acid and urates.

If then, a positive response to the Fehling and Benedict tests and, where practicable, the presence of glucosazone are used as the criteria for assessing glycosuria, it will be seen from Table II that glycosuria occurs in 6·9% of normal non-pregnant women. During the early months of pregnancy the incidence of glycosuria increases to 29·6%, and it falls to 19·8% in the middle and later parts of pregnancy.

Williams and Wills[12] have stated that glycosuria occurring early in pregnancy always persists till term. In a small series of 39 patients on whom 104 serial tests were performed, glycosuria developed in nine instances early in the pregnancy and in two during the later months. In

four instances the glycosuria shown in the early months could not be detected in the later months. This disappearance of glycosuria in nearly half of this small series of patients suggests that the statement of Williams and Wills needs modification.

Occurrence of Glycosuria after a Large Dose of Monosaccharide.

For many years glycosuria after the ingestion of large doses of glucose was regarded as an indication of pregnancy. Scheffey[6] used 120 grammes of glucose in his tests, but pointed out that the exact dosage made little difference to the incidence of glycosuria among the patients. Williams and Wills,[12] using 100 grammes of glucose, found that 60% of their patients showed glycosuria, although it occurred in only 5·4% when they were on a normal diet. Because pregnant women find difficulty in taking large doses of glucose, only 50-gramme doses were used for tests on 102 patients in this laboratory. A specimen of urine was obtained from the fasting patient, who was then given the glucose solution, and a second specimen of urine was taken one hour later. Glucose was present in fasting urine specimens from 11% of the patients in the early months of pregnancy and from 15% of those in the later months. After the ingestion of glucose the incidence of glycosuria increased to over 60% in both groups.

Table II.
Incidence of Glycosuria.

Source of Urine.	Total Number of Specimens.	Positive Reaction to Benedict's Test.	Percentage.	Positive Reaction to Fehling's Test.	Percentage.
Non-pregnant women ..	172	68	39·5	12	6·9
Women one to three months pregnant ..	340	124	36·4	101	29·6
Women three to six months pregnant ..	51	19	31·3	10	19·8
Women six to nine months pregnant ..	145	64	41·3	31	21·4

Although glycosuria is undoubtedly more common among pregnant women under ordinary conditions of diet and can be greatly increased by ingestion of large amounts of carbohydrates, this test would fail to detect pregnancy in 40% of the patients. Compared with the 98% accuracy claimed for the Aschheim-Zondek test in the diagnosis of pregnancy, the incidence of glycosuria after the ingestion of carbohydrate can hardly be regarded seriously as a test for this condition.

The Fasting Blood Sugar Level in Pregnant Women under their Normal Conditions of Diet.

The value of the fasting blood sugar level of normal, healthy subjects has been estimated to fall between 0·08% and 0·12%. Rowley[13] and Morriss[14] found that the level of the fasting blood sugar in normal pregnant women fell within the normal range. Bergsma[15] obtained a maximum value of 0·136% during the later months of pregnancy, with a mean value of 0·124%. Krebs and Briggs[16] obtained maximum and minimum values of 0·124% and 0·071% respectively. Nayar and his co-workers[17] estimated the concentration of sugar in the blood of 35 normal pregnant women and found it to be of normal average value in only four of them. The concentration of blood sugar in 23 of the remaining 31 fell below 0·07%. These figures, in which the majority of patients investigated show a low concentration of blood sugar, form a striking contrast to the results of other workers mentioned.

The fasting blood sugar level of 103 pregnant patients was estimated by the Maclean method in this laboratory. The maximum value obtained was 0·120%, the minimum value 0·040%. The average figure of 0·085% was within the range reported by most workers. However, only 33% of the patients had fasting blood sugar values of 0·09%, whilst in 24·2% it was 0·08% and in another 13·6% it was as low as 0·07%. These results indicate that an appreciable number of these pregnant women (38%) have a lowered fasting blood sugar level.

Fasting blood sugar estimations were repeated on 24 of the patients when in the non-pregnant state. The maximum value was then found to be 0·12% and the minimum 0·05%. Nineteen of these patients had blood sugar values of 0·09% and over. Therefore, comparison of the fasting blood sugar level in the same women during pregnancy and later in the normal state showed that the level was lowered in the majority of these patients during pregnancy.

The results of these investigations on the group of 103 women during pregnancy, and on 24 both during and after pregnancy, agree with those obtained by Nayar.

The Influence of Pregnancy on Glucose Tolerance Curves.

A study of the literature shows that from pregnant as well as from non-pregnant women three types of glucose tolerance curves may be obtained—namely, the normal curve, the renal glycosuric curve and a group denoting diminished carbohydrate tolerance. This last-mentioned group includes the curve termed by Maclean "renal glycosuria with superimposed diabetes", the alimentary glycosuric curve and the diabetic curve. In the "renal glycosuria with superimposed diabetes" the presence of a lowered renal threshold is revealed by the occurrence of glycosuria, although the blood sugar level never exceeds the normal threshold; but the failure of the blood sugar content to decrease to the fasting level within the two hours of the test indicates a diminution in carbohydrate tolerance.

Unless the patient is examined in both the pregnant and non-pregnant states, diminished carbohydrate tolerance cannot be said to be due to the pregnancy. Williams and Wills[12] carried out glucose tolerance tests on clinically normal pregnant women and on non-pregnant women. In non-glycosuric pregnant women they found that the average curve during the first five months of pregnancy was slightly higher than that of the normal non-pregnant patients. However, in the latter half of pregnancy the curve was slightly lower than the normal and post-confinement curve. The maximum blood sugar value reached in this group after ingestion of 100 grammes of glucose was 0·15%. In glycosuric cases they obtained two effects. Firstly, there was a low maximum blood sugar value by virtue of a lowered renal threshold, and secondly, there was a high maximum blood sugar value with a tendency towards a raised blood sugar curve. The amount of sugar excreted in most cases was negligible, varying from traces to 0·4%. After confinement no glycosuria was observed, and the curves were higher than during pregnancy. This was explained as being due to alteration in renal threshold. They found that in pregnant women with and without symptomless glycosuria the blood sugar values were lower than those obtained from the same patient in the non-pregnant state.

Tonkes[18] holds that during a glucose tolerance test on a pregnant woman the maximum value is reached much later than in the test on a normal patient, and that in all instances the blood sugar curve of a healthy pregnant woman resembles that of a mildly diabetic subject. He believes that the glycosuria present is "not of renal origin, but is alimentary". The figures given by Tonkes cover only nine cases, and it would appear that this number is not sufficiently large for any definite conclusions to be drawn.

Richardson and Bitter[19] reported that 60% of pregnant women show no gross abnormalities of sugar metabolism, and only 8% have a blood sugar curve of a type on which a diagnosis of lowered carbohydrate tolerance is usually made.

Hurwitz and Jensen[20] hold that *diabetes mellitus*, which in many instances may begin in pregnancy, may be over-

looked because of the prevalence of glycosuria at this time. They studied the effect of pregnancy on the carbohydrate metabolism of a group of 25 patients and found that the fasting blood sugar levels during pregnancy were well within the normal range. However, examination of their figures shows that most of them were in the low normal range, over half being under 0·09%. Further, the blood sugar level of their post-partum group of patients was somewhat higher. Thus their conclusion that the fasting blood sugar level is not lowered in pregnancy is not justified. The peak values of the curves obtained

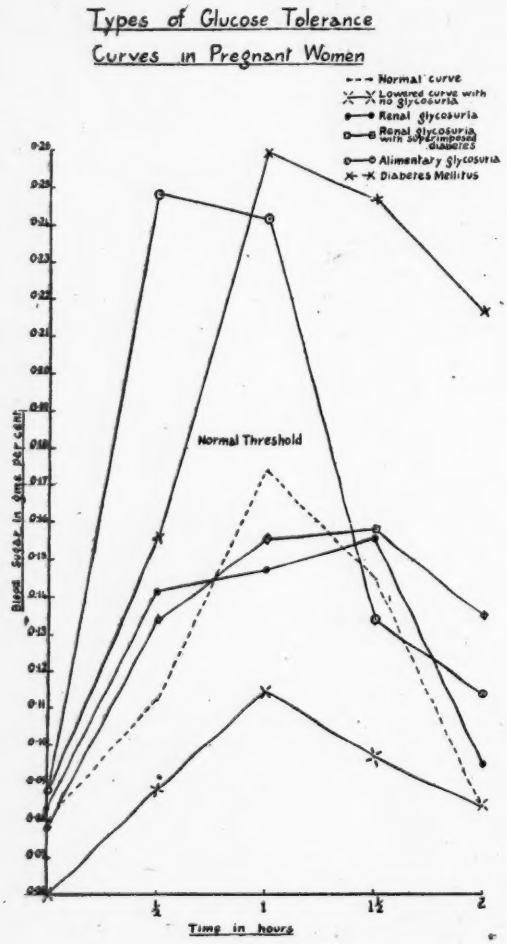

FIGURE I.
Types of glucose tolerance curves obtained from pregnant women.

by them fell within the normal limits; the two-hour levels showed a large percentage of abnormally high values during pregnancy but not in the post-partum period, and the three-hour levels were normal, both in pregnant and in post-partum patients. Hurwitz and Jensen point out that if a high two-hour level was regarded as an indication of diabetes, this diagnosis would have to be applied to 81% of the normal pregnant women of their series. Therefore, they suggest a fasting blood sugar level of more than 0·12% and a value exceeding 0·12% at the end of three hours as well as a high peak value, as criteria of diabetes. In conclusion they state that glycosuria occurring in pregnancy is due probably to alimentary hyperglycæmia rather than to a lowering of the renal threshold.

Experimental Investigation.

Glucose tolerance curves were prepared for 102 women who were attending the Women's Hospital Clinic for antenatal care. At the time of the investigation all the women were in normal health, although some of them developed toxæmia later in pregnancy.

Technique.

The patient was given instructions to have nothing to eat after 10 p.m. on the night prior to the test and to drink only water. At 9.30 a.m. on the following day specimens of blood and urine were obtained, and then 50 grammes of glucose dissolved in 150 millilitres of water were given by mouth. Specimens of blood were taken at half-hourly intervals for two hours. Urine specimens were collected at hourly intervals.

The blood sugar level was estimated by Maclean's method, and the urine specimens were examined qualitatively with Fehling and Benedict solutions. In those cases in which pronounced reduction of Fehling's solution was obtained, the amount of sugar present was estimated quantitatively by the Folin and McEllroy method.

When possible, a glucose tolerance curve was obtained in the early and later parts of pregnancy as well as twelve months after pregnancy, in order to obtain normal conditions after the lactating period was over. Great difficulty was encountered in persuading the women to return for further investigation after discharge from the hospital. In all, 170 glucose tolerance curves were prepared for 102 patients (Table III).

TABLE III.
Distribution of Glucose Tolerance Curves Obtained During the Ante-Natal and Post-Natal Periods.

Total Number of Curves.	Curves During First Five Months of Pregnancy.	Curves During Sixth to Ninth Months of Pregnancy.	Curves After Pregnancy.	Number of Exton-Rose Curves.
170	35	84	31	20

Results.

The curves obtained were classified into the following five groups, according to the type of glycosuria or its absence during pregnancy: (*a*) no glycosuria, (*b*) renal glycosuria, (*c*) renal glycosuria with superimposed diabetes, (*d*) alimentary glycosuria, (*e*) *diabetes mellitus*.

Group A: No Glycosuria.—Thirty-eight of the 102 pregnant patients excreted urine free from sugar. Thirteen curves were obtained during the first five months of pregnancy, 30 during the six to nine months' period and 13 after pregnancy. Inspection of Table IV shows that in about 50% of the patients investigated in both early and late pregnancy the maximum blood sugar value after a dose of 50 grammes of glucose is low. Table V shows that the difference between the fasting blood sugar level and the maximum blood sugar level is also decreased. However, most of these patients gave a normal response to the dose when tested twelve months after pregnancy. By then four of the patients had developed glycosuria, which appeared to be alimentary glycosuria in one instance, renal glycosuria in another, and "renal glycosuria with superimposed diabetes" in two instances.

Group B: Renal Glycosuria.—The tests on 33 patients resulted in curves which showed that the renal threshold was lowered. Eleven curves were prepared during the first five months, 29 during the six to nine months' period and 14 in the same group of patients *post partum*. The difference between the fasting and maximum blood sugar values was decreased in 40% of these patients, but showed little variation whether the patient was pregnant or not. Four of the patients had no glycosuria when tested twelve months after pregnancy. It is interesting to note (Table IV) that fewer pregnant patients in this group exhibited the low maximum values observed in Group A during the glucose tolerance test. About 60% of these patients show a normal difference between the

TABLE IV.
Peak Values in Glucose Tolerance Curves.

Pregnancy Status.	A. No Glycosuria. (Percentage of Patients.)				B. Renal Glycosuria. (Percentage of Patients.)				C. Renal Glycosuria with Superimposed Diabetes. (Percentage of Patients.)			
	0·100%	0·100% to 0·140%	0·140% to 0·160%	0·160% to 0·180%	0·100%	0·100% to 0·140%	0·140% to 0·160%	0·160% to 0·180%	0·100%	0·100% to 0·140%	0·140% to 0·160%	0·160% to 0·180%
1 to 5 months pregnant ..	—	46·0%	38·0%	15·0%	0·0%	18·2%	45·4%	27·3%	—	—	85·7%	14·3%
6 to 9 months pregnant ..	13·3%	40·0%	22·5%	20·0%	3·4%	38·0%	27·5%	31·1%	—	7·7%	23·7%	68·6%
After pregnancy	—	7·7%	23·0%	69·0%	9·1%	45·4%	18·2%	27·3%	—	—	25·0%	75·0%

fasting and maximum values in early and late pregnancy, but the difference is low in a larger number of the non-pregnant patients. These results are not unexpected, since in 24 patients the glycosuria was very slight and would not affect the blood sugar level to any degree. In twelve instances quantitative estimations of the glucose excreted during the period of the test were made, and the average amount was found to be 0·96 gramme per two hours in a series which ranged from 0·08 gramme per two hours to 4·6 grammes per two hours.

Group C: Renal Glycosuria with Superimposed Diabetes. Nineteen patients had glucose tolerance curves in which the blood sugar value failed to return to the normal fasting level within the two hours, and yet the maximum blood sugar level attained by these patients at any time during the test did not exceed 0·180%. The post-partum curves obtained in this same group were also characterized by this delay in return to fasting level. The average maximum blood sugar value was 0·14% after the dose of glucose, in both early and late pregnancy. In a small proportion of cases the difference between fasting and maximum blood sugar values appeared to be decreased.

Group D: Alimentary Glycosuria.—Ten raised curves which resembled the Maclean lag curve, with peak values all exceeding 0·180%, were obtained from seven patients. Three such curves were obtained in the early months and seven in the latter period of pregnancy. In the one case in which a follow-up curve was obtained during pregnancy, the maximum value fell from 0·220% in the early period to 0·171% in the latter part. The amount of sugar excreted varied between traces and 0·15 gramme per two hours. The difference between fasting and maximum blood sugar levels was in all cases either normal or slightly higher than normal.

Group E: Diabetes Mellitus.—Only five patients in the total of 102 had diabetic curves, giving an incidence for diabetes of 4·9%; but this figure cannot be taken as the normal incidence of diabetes in pregnancy, since these diabetic patients were deliberately included in this investigation.

THE TWO-DOSE GLUCOSE TOLERANCE TEST.

The two-dose, one-hour glucose tolerance test, first advocated by Exton and Rose in 1934, has been used by Wayburn and Gray,[23] by Langner and Dewees[23] and by others to overcome the fluctuations caused in the one-dose test by the previous diet of the patient.

In the two-dose, one-hour glucose tolerance test fasting specimens of blood and urine are taken and then the patient is given a dose of 50 grammes of glucose. Half an hour later further specimens are taken and the patient is given a second dose of glucose. One hour after the beginning of the test final samples of blood and urine are obtained.

According to Exton and Rose, three criteria are necessary for assessing a normal response—namely, (i) a normal fasting blood sugar value, (ii) a rise in the blood sugar level which does not exceed 0·075% in the half-hour specimen, and (iii) a one-hour blood sugar value which does not exceed the half-hour value by 0·010%.

Many workers have agreed that this interpretation, originally given by Exton and Rose, is too rigid, as it would label many normal individuals as diabetics. Wayburn and Gray[21] interpret a rise in the blood sugar level of the order of 0·158% to 0·179% at the end of one hour as being doubtful, and a rise of 0·180% to 0·260% as indicating mild diabetes, and they diagnose the patient as severely diabetic when this value is exceeded.

Langner and Dewees state that the two-dose, one-hour test is very sensitive, and suggest that it should be used as an exclusion test in borderline cases, but that the result should be rechecked by the one-dose test. Mathews and his co-workers[25] emphasize that if the peak value after the second dose does not exceed 0·160% the response is normal. Gould[24] suggests a total rise of not more than 0·080%.

With the object of obtaining more information regarding possible derangement of carbohydrate metabolism than is obtainable by the one-dose, two-hour glucose tolerance curve (Maclean), the Exton-Rose two-dose, one-hour test was also performed on 20 pregnant patients. However, the test was extended to cover two hours. All the blood sugar estimations were performed in duplicate. A considerable amount of difficulty was encountered in persuading the patients to drink the second dose of glucose, as they developed nausea and in some cases vomiting.

Two of three patients, who were found to be normal when tested by the Maclean method, also gave normal

TABLE V.
Difference Between Fasting Blood Sugar Level and Maximum Blood Sugar Level.

Pregnancy Status.	A. No Glycosuria. (Percentage of Patients.)				B. Renal Glycosuria. (Percentage of Patients.)				C. Renal Glycosuria with Superimposed Diabetes. (Percentage of Patients.)			
	0·040%.	0·040% to 0·060%.	0·060% to 0·080%.	0·080% to 0·100%. (Normal Range.)	0·040%.	0·040% to 0·060%.	0·060% to 0·080%.	0·080% to 0·100%. (Normal Range.)	0·040%.	0·040% to 0·060%.	0·060% to 0·080%.	0·080% to 0·100%. (Normal Range.)
1 to 5 months	7·7%	53·8%	30·3%	7·7%	9·0%	27·3%	18·2%	45·4%	—	14·3%	57·1%	28·6%
6 to 9 months	20·0%	40·0%	20·0%	20·0%	6·2%	27·5%	27·5%	37·9%	—	30·7%	30·7%	38·6%
After pregnancy	7·7%	15·3%	38·4%	38·4%	21·5%	35·7%	28·5%	15·0%	—	—	25·0%	75·0%

responses to the Exton-Rose test; but the third patient developed renal glycosuria in response to the extra dose of glucose. Of the five patients assessed as having renal glycosuria by both tests, one may also be said to be mildly diabetic according to the Wayburn and Gray interpretation of the Exton-Rose test. The Maclean test suggested that seven patients had "renal glycosuria with superimposed diabetes". In four of these instances a similar diagnosis of doubtful diabetes would be made from the results of the Exton-Rose tests; but in the other three instances this test failed to indicate the diabetic tendency suggested by the Maclean test, and these patients appeared to have nothing more than renal glycosuria. Among the remaining five patients in the series three were assessed as having alimentary glycosuria, one as having a delay in the capacity to store glucose, and the fifth as mildly diabetic by the Maclean test, whilst all were assessed as mildly diabetic by the Exton-Rose test.

In the Exton-Rose test, when insufficient time is allowed to see whether the blood sugar value returns to the fasting level in the normal time, a patient having alimentary glycosuria would be considered to be mildly diabetic. This seems to be the main fallacy in the Exton-Rose test.

In this small series as much information has been obtained by the Maclean test as by the Exton-Rose test, and the danger is shown of classifying alimentary glycosurics as mildly diabetic if the Exton-Rose test only is used. In those cases in which a doubtful result is obtained it is necessary to carry out further tests on the patients and follow up the clinical condition over a considerable period before one can determine which of these two tests gave the true diagnosis. No severe abnormal condition could be found in any of the patients during the period over which tests were carried out.

In cases in which there is definite clinical evidence of diabetes the preparation of a single glucose tolerance curve is all that is necessary to confirm the diagnosis. If only slight glycosuria is present, and if there is little in the way of clinical symptoms, a single glucose tolerance curve may be misleading. Repetition of the test under normal conditions of diet and relief from mental stress is desirable. Corkill and Marks[26] have shown that emotion, mental stress and worry may provoke glycosuria in some otherwise normal subjects. If desired, the Exton-Rose test may be used as a confirmatory test.

It is evident that if the patient is known to have been taking a diet rich in fat or poor in carbohydrate, a normal diet should be resumed for several days before she is referred to the laboratory for investigation. From the evidence presented it seems that the chief value of the Exton-Rose test is as a confirmatory test in those cases in which the result of the Maclean test is difficult to interpret. This will occur only in those cases in which the diabetic condition is still very slight or in which some extraneous factor is the cause of some abnormality in the curve. There is not sufficient evidence as a result of this investigation to support the claim that the Exton-Rose test must be regarded as representing the final diagnosis when differences between the results of the two tests occur.

DISCUSSION.

From the results presented here, the often-expressed idea that carbohydrate metabolism is altered during pregnancy is confirmed. An increase occurs in the excretion of glucose by some pregnant women receiving a normal diet and by the majority of women after the ingestion of a large amount of monosaccharide. The incidence of glycosuria in normal non-pregnant women was found to be 6·9%, and in pregnancy approximately 20%, increasing to 60% after a dose of 50 grammes of glucose. This agrees with the work of Williams and Wills, who found 5·4%

TABLE VI.

Comparison of the Results of the Maclean and Exton-Rose Glucose Tolerance Tests.[1]

Subject Number.	Maclean Glucose Tolerance Test. (Grammes *per Centum.*)	Comment.	Exton-Rose Test. (Grammes *per Centum.*)	Increase in Blood Sugar from Fasting to One-half Hour. (Grammes *per Centum.*)	Increase in One Hour. (Grammes *per Centum.*)	Interpretation of Exton-Rose Test by Wayburn Gray Standards.
I	0·095, 0·135, 0·151, 0·093 (no glycosuria).	Normal.	0·076, 0·136, 0·153, 0·158	0·060	0·077	Normal.
II	0·092, 0·141, 0·135, 0·095, 0·089 (no glycosuria).	Normal.	0·093, 0·146, 0·146, 0·135	0·053	0·053	Renal glycosuria.
III	0·113, 0·150, 0·194, 0·155, 0·121 (no glycosuria)	Normal with raised threshold.	0·077, 0·134, 0·133, 0·104 (no glycosuria).	0·057	0·057	Normal.
IV	0·092, 0·109, 0·145, 0·121, 0·116	Renal glycosuria.	0·084, 0·100, 0·134, 0·122	0·016	0·050	Renal glycosuria.
V	0·083, 0·134, 0·117, 0·106, 0·092	Renal glycosuria.	0·112, 0·114, 0·125, 0·136	0·002	0·013	Renal glycosuria.
VI	0·085, 0·099, 0·099, 0·130, 0·121	Renal glycosuria.	0·088, 0·135, 0·125, 0·122	0·047	0·037	Renal glycosuria.
VII	0·078, 0·136, 0·135, 0·104, 0·127	Renal glycosuria.	0·075, 0·131, 0·159, 0·143	0·056	0·084	Renal glycosuria; ? mild diabetes.
VIII	0·092, 0·109, 0·185, 0·151, 0·117	Renal glycosuria.	0·075, 0·134, 0·156, 0·118	0·059	0·081	Renal glycosuria.
IX	0·085, 0·151, 0·161, 0·170, 0·166	Renal glycosuria with superimposed diabetes.	0·077, 0·116, 0·158, 0·133	0·039	0·081	Renal glycosuria.
X	0·079, 0·134, 0·156, 0·158, 0·135	Renal glycosuria with superimposed diabetes.	9·096, 0·143, 0·170	0·047	0·074	Renal glycosuria; doubtful diabetes.
XI	0·119, 0·176, 0·135, 0·147	Renal glycosuria with superimposed diabetes.	0·096, 0·133, 0·171, 0·158	0·037	0·075	Renal glycosuria; doubtful diabetes.
XII	0·080, 0·149, 0·176, 0·132	Renal glycosuria with superimposed diabetes.	0·080, 0·134, 0·133, 0·177	0·054	0·054	Renal glycosuria.
XIII	0·061, 0·127, 0·134, 0·130, 0·132	Renal glycosuria with superimposed diabetes.	0·093, 0·160, 0·167, 0·153	0·067	0·074	Renal glycosuria; doubtful diabetes.
XIV	0·075, 0·112, 0·147, 0·153, 0·130	Renal glycosuria with superimposed diabetes.	0·061, 0·121, 0·168, 0·155	0·060	0·107	Renal glycosuria; doubtful diabetes.
XV	0·104, 0·121, 0·152, 0·180, 0·173	Renal glycosuria wth superimposed diabetes.	0·075, 0·111, 0·127	0·036	0·052	Renal glycosuria.
XVI	0·085, 0·158, 0·162, 0·135, 0·134 (no glycosuria).	Delayed storage.	0·084, 0·143, 0·182, 0·156	0·059	0·098	Mild diabetes.
XVII	0·079, 0·206, 0·192, 0·152, 0·129	Alimentary glycosuria.	0·085, 0·184, 0·221, 0·184	0·099	0·136	Mild diabetes.
XVIII	0·101, 0·206, 0·199, 0·220, 0·118	Alimentary glycosuria.	0·095, 0·168, 0·166, 0·134	0·075	0·075	Mild diabetes.
XIX	0·098, 0·209, 0·187, 0·152, 0·117	Alimentary glycosuria.	0·100, 0·143, 0·184, 0·132	0·043	0·084	Mild diabetes.
XX	0·090, 0·155, 0·155, 0·185, 0·157	Mild diabetes.	0·092, 0·147, 0·203, 0·182	0·055	0·111	Mild diabetes.

[1] Unless otherwise stated, glycosuria was present.

glycosuria among normal patients, increasing to 60% among pregnant patients after a 100-gramme dose of glucose. Their statement that glycosuria detected early in pregnancy always persisted until term could not be substantiated.

In view of this high percentage of pregnant women who developed glycosuria, to act on Eastman's[26] statement that glycosuria discovered during pregnancy should be treated as *diabetes mellitus* until proved otherwise would not be advisable, since any attempt to make the urine sugar-free through the administration of insulin might lead to severe ketosis. The glycosuria during the early months of pregnancy, termed "early alimentary glycosuria" by Lambie,[27] and said to be due to a decrease in assimilation as well as to a lowering of the renal threshold, is attributed by Herold[28] to an over-activity of the sympathetic nervous system and its associated glands.

Host[29] considers that it is due to a decreased tolerance for carbohydrate, whilst Richardson and Bitter suggest the possibility of damage to the kidney tubules. This seems unlikely, since Friedman and his co-workers[30] have shown that true renal glycosuria is not associated with renal disease or with glomerular dysfunction.

Lambie suggests that late alimentary glycosuria may be due to transient diabetes of pregnancy, which can lead to permanent diabetes. Host maintains that a lowering of the renal threshold is the cause of glycosuria in the latter half of pregnancy.

The idea of excessive mobilization of glucose during pregnancy suggested by Ehrenfest[31] to explain the occurrence of glycosuria in pregnancy does not seem to be borne out by these observations, since from only four patients other than the diabetic mothers was a raised blood sugar curve obtained. These results are also in disagreement with those of Williams and Wills and of Tonkes, who hold that glycosuria in pregnancy is due more to a raised blood sugar level than to a lowering of the renal threshold. Only four glycosuric patients with blood sugar curves of this type were discovered in my group of 102 patients, and there was no evidence to show that the renal threshold was altered. One group of glycosuric patients did have a lowered threshold and showed what Maclean terms a "superimposed diabetes". This was revealed by the failure of the blood sugar to return to fasting level about two hours after the dose of glucose, showing that there was a delay in the ability to store glucose as glycogen. However, in these cases the effect of the glycosuria was to depress the whole curve so that the peak value never reached the normal threshold value of 0·18%. As has been previously pointed out, many of these patients yielded exactly the same curve when tested in the non-pregnant state, so that the pregnancy could not be the cause of the glycosuria.

In these investigations the glycosuria detected at any time during pregnancy was seldom great enough to be responsible for the loss of large quantities of glucose, and therefore it failed to explain the lowered glucose tolerance curves obtained from so many patients. More interesting still is the fact that this particular type of glucose tolerance curve was obtained from half the patients who showed no glycosuria. Such curves have not been reported by other workers; but Laferty[32] has suggested four conditions operating in pregnancy which could cause disturbances in carbohydrate metabolism and thereby explain this lowered glucose tolerance curve, namely: (i) the continuance of heavy withdrawal of glucose from the maternal blood to nourish the products of conception; (ii) an excessive consumption of glucose by the maternal organism; (iii) the excessive withdrawal of glucose from the maternal blood due to over-production of insulin; (iv) an inadequate supply of glucose due to faulty alimentation.

These factors, either singly or in combination, would seem to explain the lowered curves obtained from non-glycosuric patients. The fact that so many pregnant women appear to be in a state in which carbohydrate either is not assimilated or is being rapidly removed from the blood stream is particularly important in cases in which the restriction of carbohydrate may be contemplated. Such action may precipitate a condition of severe acidosis.

SUMMARY.

The evidence presented as a result of this investigation has confirmed some claims with regard to certain aspects of carbohydrate metabolism in pregnancy and has contradicted others.

1. Symptomless glycosuria is present in about 30% of women during early pregnancy and in about 20% in the later months, when the patient is receiving her normal diet.

2. Many pregnant women are unable to deal normally with large doses of monosaccharides taken at one period. There is an increased incidence of glycosuria after a dose of 50 grammes of glucose; 60% of pregnant women exhibited this reaction.

3. Glycosuria observed early in pregnancy need not persist throughout it.

4. A low fasting blood sugar level occurs in many pregnant women.

5. The incidence of true *diabetes mellitus* in pregnant women is low.

6. Except in a few instances of alimentary glycosuria the glycosuria of pregnancy seems to be due to a lowering of the renal threshold, and usually this is not associated with a raised blood sugar level. The glycosuria was generally responsible for excretion of relatively small amounts of glucose, and except in a few instances would not account for the lowered curves obtained from these patients.

7. The most interesting new fact disclosed by these investigations was the lowering of the glucose tolerance curve of about half of the patients who exhibited no glycosuria after the ingestion of 50 grammes of glucose.

8. The value of the Exton-Rose test lies mainly in its use as a confirmatory test in those cases in which the result of the Maclean test is difficult to interpret.

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Reviews.

PRACTICAL BIOLOGY.

THE publication of textbooks especially written for medical students is of particular interest in these days of reconstruction when so few books are available and classes are large, and this applies even to an elementary textbook. One looks at new books, or new editions, for some indication of new trends in teaching, or new ideas regarding syllabuses. The publication of a second edition of "Practical Biology for Medical and Intermediate Students", by C. J. Wallis, is of interest from both these viewpoints.[1]

Unfortunately a practical textbook for biology designed for use in Great Britain is of far less use to Australian students than textbooks in any other science. The content of a practical syllabus in chemistry, physics or human anatomy in London might be identical with one used in Sydney. This could never be the case in biology, since Australian classes must use local types and material. The book under review, however, is more useful than most of its predecessors, for it includes sections on microscopic technique and elementary biochemistry in addition to botany and zoology. Sections on animal cytology, histology and animal physiology *et cetera* are also included.

The book is one of those practical handbooks which are illustrated and the figures are clear and numerous. Some of the histological diagrams are perhaps a little too sketchy. Others are adequate. One might say that for a medical student just beginning in a large practical class in Great Britain the volume would be a useful, indeed a valuable, guide. The proofs have certainly been through the hands of some very experienced teachers.

Besides the grouping of botany and zoology sections in one book, and the sections on biochemical technique, one very good point is certainly the inclusion of many simple experiments. The experimental and unified outlook is welcome. Despite this, however, we must confess to a feeling of disappointment. We seem to be remarkably close to the textbooks of thirty and more years ago, especially in the part devoted to animal biology. And there is little doubt that this is a reflection of many of the present university syllabuses in Great Britain; indeed the author leaves no doubt about this on the title page.

The first question which arises is whether first year biology for medical, intermediate science and higher school certificate students should be the same. The medical students are going to devote years to the study of man. Many of the others will never do any more biology at all. It is doubtful on this test whether the course provided is the best practical introduction for such students. Something could certainly be said, too, for the addition of some elementary work in genetics. But perhaps this is very unfair to the author whilst the authorities planning university and medical school curricula remain so closely tied up in the traditions of the past.

For more than one reason (in particular the necessity for an introduction to genetics in the corresponding theory or lecture courses) the practical work on cytology is of considerable importance to medical students. This section is, however, weak, and scarcely up to date. Room for improvement thus remains. In many respects it still seems more difficult to produce introductory works than advanced textbooks.

The printing and paper are good and approach closely British pre-war standards. There is a satisfactory index.

[1] "Practical Biology for Medical and Intermediate Students", by C. J. Wallis, M.A. (Cantab.); Second Edition Revised, Enlarged and Reset; 1947. London: William Heinemann (Medical Books), Limited. 8½" × 5½", pp. 406, with many illustrations. Price: 21s.

THE PERSONALITY OF THE PRE-SCHOOL CHILD.

IN "The Personality of the Preschool Child" Dr. Werner Wolff has attempted to analyse the behaviour of young children in their spontaneous verbal, graphic and dramatic expression.[1] His entire philosophy merges round one theme—the child's search for his self and his subsequent development of that self. He emphasizes repeatedly the view that the young child and the adult live in worlds apart.

Unlike most researchers in this field of child personality he maintains that the child's thought has every appearance of being exclusively unrealistic. Whether we agree with this or not, Dr. Wolff has presented us with a considerable amount of interesting data which he has submitted in a convincing manner.

He has once again raised the very controversial subject of the place of fairy tales in the development of the pre-school child, and in his support of them he will find many psychologists and teachers to agree with him.

His theory that rhythmic quotients are generally more reliable and therefore more desirable than intelligence tests is a new one to Australian pre-school teachers and child psychologists, and will probably cause considerable debate and experimentation before it will be accepted as a standard means of assessing child personality.

The author outlines the main features used in graphoanalysis and gives a series of personality sketches based on consideration of the child's scribblings. These are subsequently checked by the case history and show a high degree of correlation.

As research associate in the Department of Child Study at Vassar College, New York, and as professor of psychology of Bard College, Dr. Wolff has had exceptional opportunities to observe development in this field and his interpretation of children's personality deviations gives us material for considerable thought. The book makes agreeable reading and has an extensive bibliography. It is worth perusal by all who are interested in the development of personality.

[1] "The Personality of the Preschool Child: The Child's Search for His Self", by Werner Wolff; 1947. London: William Heinemann (Medical Books), Limited. 9" × 6", pp. 358, with illustrations. Price: 25s.

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THE EPIC OF MEDICINE.

PROFESSOR CHAUNCEY D. LEAKE, of the Medical School of Texas University, well known not only as a pharmacologist of world repute, but also for his contributions to general culture, including a new translation of William Harvey's *De Motu Cordis*, has lately annexed the province of poetry and has published in free verse a metrical exposition of the discovery and progress of anæsthesia entitled *Letheon*. His model has been to some extent Stephen Vincent Benét, whose deservedly praised *John Brown's Body* followed the lead of Hardy's *Dynasts*, and in the opinion of many manifests a more sustained flight of diction. Though the stage which Benét chose for his dramatic creation was far smaller than the Europe of the Napoleonic wars, yet the period he selected was able to give him excellent opportunities for the portraiture of strong characters such as Lincoln, Lee, "Stonewall" Jackson and other leaders in the American Civil War. Leake's theatre of action is smaller still and the personalities have little of the heroic in them, but nevertheless there are incidents of truly dramatic complexion. He begins with the early and crude attempts to stupefy or nauseate the patient as with mandragora; then, of course, comes Humphry Davy with his "laughing gas", and by the way Humphry Davy is worth more vigorous treatment not only in the realm of discovery but in personality, for Davy was a man of most attractive appearance and address. Then we have the unhappy Wells and Morton episode; then Simpson strongly advocating chloroform and adroitly rebuffing the charge that in deadening the pains of childbirth he was opposing scripture. And so the story goes on, told by one whose scholarship in this particular department of medical history is admittedly in the front rank. Professor Leake is certainly to be congratulated on his pioneer labours in finding material for poetry in this or indeed any branch of medical science. Others, we trust, may follow his example and give the world in general an inspiring description of the march of discovery in any of the many fields of medicine and surgery where dramatic incidents assuredly abound. Naomi Jacobs has recently told in fiction, with a breadth and arresting force reminding one of Victor Hugo, the sombre history of rubber. Why has no one thought of the history of quinine which in many ways would be more acceptable. Naomi Jacobs's forceful pages depict greed, fraud, callous upholding of vested interests and horrible cruelty and oppression, whereas the story of quinine, and it possesses many epic qualities, is almost free from these black and forbidding clouds? The saga of quinine might begin with the South American Indians, then the Jesuit missionaries, then the picturesque quack Talbor, and would end with Charles Ledger, of Goulburn, New South Wales, who was really the man who gave quinine to the world and after whom the plant yielding the highest amount of the alkaloid in the Dutch East Indies has been appropriately named *Cinchona ledgeriana*.

Kipling has given us the dour Scots engineer M'Andrew, realistically efficient in his mechanical duties, but proudly and to some degree romantically attached to his big reciprocating steam engines in the ocean liner and bewailing the fact that the objects of his affection have not attracted the notice of a true poet:

> I'm sick of all their quirks an' turns . . . the loves and doves they dream . . .
> Lord, send a man like Robbie Burns to sing the Song o' Steam.
> Oh, for a man to weld it then, in one trip-hammer strain,
> Till even first-class passengers could tell the meanin' plain.

Kipling, though untrained in engineering, all the same came close to fulfilling M'Andrew's desire, for the poem is counted amongst his best. But if steam lacked a laureate, what must we say concerning the poetic silence which enshrouds the long narrative of medicine and surgery? The great epics of the world have not always dealt with war, though certainly there has been war in superfluity, such as the siege of Troy, the exploits of Roland Paladin, the plundering forays of the Vikings or the conquest of Jerusalem in the First Crusade. Even in Milton the revolt and defeat of Satan and his powers of darkness fill most of the enormous theatre of action in "Paradise Lost". But on the other hand the Lusiads of Camoens were inspired by the marvellous voyage of Vasco da Gama, and the *Divina Commedia* of Dante presents a terrible picture of sin and its punishment with a subsequent glowing and radiant description of the admission of the virtuous to the realms of the blessed. The long, involved annals of medicine reveal many an episode of intense dramatic quality and many a personality of commanding greatness. Even the mere nomenclature of the parts of the body has had impressed upon it the labours of Greek, Roman, Byzantine, Hebrew, Moor, mediæval physician and Renaissance scholar. Our own race has not been backward in providing both towering personality and brilliant achievement in the healing art. William Harvey, Thomas Sydenham, John Hunter and Joseph Lister are names of magic import and their works have followed them. There is abundant material here for the poet, dramatist and novelist to sort and to assimilate and to breathe into it the spirit of life by creative artistry, Chauncey Leake has taken one isolated record out of the vast and elaborate archives of medical history, and his choice we may imagine has been dictated chiefly by his training and research in pharmacology; but we may hazard the guess that the fact of the pioneers of anæsthesia, Wells and Morton, being fellow countrymen, has not been with-

out influence. It has been said of the doctor that when he writes well he writes very well, but this appraisement pertains rather to prose. The contribution of medical men to literature has been critically examined by more than one writer, not the least being Dr. Boyd Graham, who published his survey in this journal; but so far medical authors have adopted traditional themes and their works have been little coloured by their profession, the poems of the laureate Dr. Robert Bridges being a case in point. Oliver Wendell Holmes wrote two "medicated novels", as they were termed, and the reading public did not like them. An extended field awaits the poet who can write with directness and simple force, not trying to invent overstrained metaphors, one who avoids the antics of certain so-called moderns at which the angels will not bother either to laugh or to weep.

Current Comment.

ROYAL PHYSICIANS AND THE DEATHS OF KINGS.

IT seems to be a matter of considerable satisfaction to many modern historians that their histories are no longer written with kings and battles—especially kings—as the main landmarks. They present a much more comprehensive and unbiased account by concentrating on essentials, the only difficulty being the decision as to what are essentials. However, whatever the merits or demerits of the lives of kings as a basis of history, it seems rather a pity that more attention has not been paid to their deaths—not, that is, to ensuring that they did die, for many earnest and assiduous people have from time to time devoted themselves to this with great efficiency, but to recording the manner and circumstances of their deaths. For a good deal can be learnt from these details. Naked facts of social, economic and political life are often seen as the personal influence of the monarch fades and his mortality yields to factors of life shared with his subjects. Most revealing of all may be the picture presented of medical knowledge and custom. Not all kings have laid their heads, as Arthur is said to have done, in the lap of a queen, "the tallest of them all and fairest", and moved in stately barge "to the island-valley of Avilion". In most cases, as F. T. Gardner[1] has reminded us, "the mighty of the past, though surrounded by voluminous soiled velvet and many dented gold vessels, still writhed on the same old bed of pain". Gardner, who presents us with the results of his inquiry into the death of William the Conqueror, complains that William's medical attendants seem to have observed carefully every detail of his final illness except the medical ones, and, in many cases, it is not easy to sort the wheat from the chaff of royal physicians' records. Reference to that memorable history of England "1066 And All That" makes it clear that "surfeits" have been particularly lethal to English kings; from the time when Richard the First succumbed to "a surfeit of Saladins" and John to a "surfeit of peaches and no cyder" to Victoria's demise from "a surfeit of Jamborees, Jokes, Gladstone, etc.", royal surfeits seem to have repeatedly baffled medical attendants, which may account for some of the curious records extant.

William the Conqueror, Gardner suggests, may have suffered from diverticulitis of the colon. An ingenious picture is built up of William's hygienic background: his active, rather violent life; his seasonal diet—meat in plenty during summer with loaves or pudding of roughly ground flour (retaining its vitamins), fruit from his garden and not too many vegetables, followed in the winter by a grossly inadequate fare that would have produced an "annual touch of scurvy"; a probable tendency to constipation from a combination of unsatisfactory diet, winter restrictions on his customary exercise and the lack of reasonable hygienic arrangements about the castle (the last not being meant to suggest squeamishness but that "icy cold castles can force a certain added reluctance on a sluggish colon"). In his fifties the previously robust William grew excessively corpulent and this was given as the reason that he was brought to his bed, in Rouen, at about the age of fifty-eight years. By a process of elimination Gardner puts the blame on diverticulitis of the colon—perhaps a "presumptuous" diagnosis, "but his age, his vague distress, his grossly fat body, and his probable chronic constipation clamor their acceptance of the condition".

The prolonged illness evoked from William's cousin, Philip of France, the remark that he was surprised "that his Brother England should be so long a time in being delivered of his big belly". William maintained the rude metaphor by retorting that "at his churching [an essentially puerperal ceremony] he would set all France ablaze", and his outraged masculinity drove him onto his feet. Reviving an old quarrel as a pretext, he attacked Mantes, besieged and took it. While his troops enthusiastically carried out his instructions to scatter, pillage and burn, "the huge, sick, old man rode everywhere shouting and scowling". His horse trod on a cinder, the startled leap flung William against the pommel of the saddle and something happened inside him. Brought back to Rouen by litter, he received the attention of his physicians, Gilbert Maminot, Bishop of Lisieux, and Goutard, Abbot of Jumièges, "with some others well skilled in medicine", but though they record in detail his conversations, the making of bequests and their prolonged religious ministrations, they have left not one clinical note of value. William lay in agony for four weeks. Gardner surmises that

> the blow from the saddle, while it did not rupture the distended sac of one or another of his chronic diverticula, evidently caused such injury to the walls that it slowly necrosed. He must have become more and more toxic and the sac more and more inflamed until, during the night of the 8 or 9th of September, it ruptured at last, and peritonitis intervened.

The Conqueror had a few hours of quiet slumber and, in the early morning of September 9, 1087, died.

Immediately his devoted watchers vanished to claim their bequests; the body servants stripped the body, searched the room, turned out the bed, and fled; not till three hours later did anyone return to pick up the remains of the discarded king. Only after much difficulty was someone found to provide the funds necessary to transport the body to Caen for burial in the Abbey of Saint Stephen as ordered by the Archbishop of Rouen. The obsequies were twice interrupted—once by a nearby fire which everyone ran to fight, and once by a certain Ascelin Fitz-Arthur who objected to William's burial there on the grounds that William had wrested the land by force from his (Fitz-Arthur's) father, but fortunately "his injured heart responded to the sound of silver". The service finally over, the body was conveyed to the stone coffin, which was found to be too small. "There was nothing to do but force things a little bit"—a little bit that was just too much.

> The conditions of death, the time element, the inept embalming, and now this violent compression, conspired to put a final strain on William's abdominal wall. It burst.
>
> As the stench filled the church the last shred of dignity left the obsequies. The lid of the coffin was clapped on hurriedly and, once more, people poured from St. Stephens. William lay alone as he would lie until the sacking of the church 500 years later when his bones were disinterred and rudely treated. At that time it was discovered that, at least, he had been given one royal touch. His body was found wrapped in crimson taffeta.

Though Gardner laments the absence of clinical record, the apparent neglect of William's physical needs by his physicians may not have been to his disadvantage; it is improbable that they would have brought comfort.

[1] *Surgery, Gynecology and Obstetrics*, April, 1948.

William's was a particularly unenlightened age medically, and even six hundred years later England's king suffered much at the hands of his physicians. Charles II, so Howard W. Haggard tells us in "Devils, Drugs and Doctors", was being shaved on the morning of February 2, 1685, when with a sudden cry he fell backward and had a violent convulsion. He became unconscious, rallied once or twice, and after a few days died. Small wonder, for though we may readily posit a cerebral embolus or other essential cause of death, the royal physicians undoubtedly speeded the king's departure. Without delay after his collapse they bled him of one pint and then cupped eight ounces from his shoulder. An emetic followed with two rapidly succeeding purgatives; then an enema containing fourteen ingredients including antimony, rock salt and all the resources of the kitchen garden, repeated in two hours with another purgative. The royal head was shaved and a blister raised on the scalp. Then came a sneezing powder of hellebore root, a powder of cowslip flowers "to strengthen his brain", frequent cathartics interspersed with soothing drinks of barley water, licorice and sweet almonds, white wine, absinthe and anise, extracts of thistle leaves, mint, rue and angelica. A plaster of Burgundy pitch and pigeon dung was applied to the king's feet and he was again bled and purged. Melon seeds, manna, slippery elm, black cherry water, extract of flowers of lime, lily-of-the-valley, peony, lavender, dissolved pearls, gentian root, nutmeg, quinine and cloves—all failed to effect a cure, and forty drops of extract of human skull were given "to allay convulsions". Raleigh's antidote, which contained "an enormous number of herbs and animal extracts", was followed by bezoar stone. The physicians despaired, but were determined not to be inactive. "As a sort of grand summary to this pharmaceutical debauch", Haggard states, "a mixture of Raleigh's antidote, pearl julep and ammonia was forced down the throat of the dying king". And so after four days of intensive therapy Charles most ungratefully died.

James I was rather more canny than his grandson, and, though he suffered from many ills in his lifetime, abjured physicians. True, he knighted the Royal Physician, but he took physic only once and refused to be bled; he died "bravely resisting medical treatment". It is difficult to deny his wisdom, even though there were men of high intelligence and scientific outlook in the medical profession. The deathbed of Henry II of France brought together two of the really great, Vesalius and Ambroise Paré. Fragments of his opponent's lance had entered the king's head when the lance was splintered against his orbit in a joust. Paré and Vesalius experimented on the heads of six executed criminals in an unsuccessful effort to determine the course of the splinters. The king died after eleven days, seemingly being spared the concentrated therapy that he might have received from more attentive physicians, and Paré, in a matter-of-fact manner not unworthy of present-day pathology, described as the cause of death a brain abscess found at autopsy. The same scientific approach marks Paré's account of the death of the King of Navarre, who sustained a fatal wound of the shoulder, with a retained bullet, at the battle of Rouen.

By 1821, when the Emperor Napoleon died on St. Helena, morbid anatomy was better appreciated, but clinical treatment still tended to combine vigour with futility. Esther H. Vincent[1] has drawn a striking picture of Napoleon and the influence of his medical condition on history. With a remarkable family history of gastric cancer and a vehement unwillingness to admit to others the possibility of its existence in himself, he yet finally succumbed to it. He was convinced in his own heart (as he admitted when dying) that he was fated to die from cancer of the stomach, and so believed that he had a charmed life in battle—a fact that throws some light on his apparent recklessness and disregard of danger in the face of the enemy. Napoleon was much interested in medical matters and showed a deal of discrimination in his choice of medical men and in his encouragement of such procedures as vaccination. However, he was a difficult patient, believing in "self-regulation" and saying that he preferred to die from disease than from medicine. On St. Helena he was far from happy; to the burden of his epigastric pain and vomiting were added the bullying tactics of the governor, a diet that promoted scurvy and the ministrations of a well-meaning doctor who considered the Emperor to be suffering from hepatitis and plied him with purgatives. When this doctor departed, Napoleon was left alone for some months, until finally a young Corsican, Francesco Antommarchi, professor of anatomy at Florence, was sent to look after him. Antommarchi, however, regarded the illness as imaginary and provided little help. He laughed at the dying captive and put tartar emetic in his lemonade. By the time another doctor, an army surgeon named Archibald Arnott, was called in, Napoleon was having gastric hæmorrhages and his heart action was very weak. Arnott recognized the source of the trouble, but Antommarchi's response to the new diagnosis was the application of a vesicatory to the epigastric region. Napoleon went down hill rapidly and died in the early morning of May 4, 1821. The autopsy, performed by Antommarchi in the presence of Arnott and six other army surgeons, revealed a cancerous mass involving almost the entire internal surface of the stomach and all adjacent lymph glands.

The loneliness of monarchs is proverbial and in personal matters they seem often to have stood quite defenceless before those near to them, victims of any who would work them good or ill. When Death lays his icy hand on kings, sceptre and crown indeed must tumble down, but the icy hand has often brought the touch of benison, for with it came release from the devoted care of royal physicians and all who would do them good.

[1] *Surgery, Gynecology and Obstetrics*, January, 1948.

VOLUNTARY CONTROL OF THE HEART.

THE capacity to accelerate the heart's action voluntarily is not a new phenomenon. It is said to have been observed among certain of the devotees of Eastern systems who practise various forms of bodily control, and H. Feil, H. D. Green and D. Eiber have recently reported a case[1] which is, they state, at least the twenty-first in the literature. There are, however, points of special interest. The subject, a young medical student, tried voluntarily to dilate his pupils by using the principle of the fear response. He decided to recall an emotion of fear, a frightening nightmare experienced four years previously when he was lost in the Canadian wilds. He wrote out his experiences and re-enacted them in his imagination. This experience was practised daily for four weeks, with intense concentration on the climax of the story. He observed the dilation of his pupils in a mirror and noted the accompanying sensations, which included tachycardia. After repeated trial he was able to dissociate the effort to dilate his pupils and to concentrate on the will to accelerate his heart and found that he was successful. After an interval of a year he found again that he was able to control his heart rate at will. Upon concentration and the sudden acceleration of the heart he felt as if subjected to an explosion, and had paræsthesia of the hands and feet, tinnitus and palpitation. On willing his heart to go slow he felt relaxed and had a sensation of relief and of fatigue. He frequently felt dissociated from his environment. Euphoria and a sense of numbness often followed. In addition to this voluntary phenomenon and unassociated with it, this student had attacks of paroxysmal tachycardia five or six times a year, lasting two or three hours. No physical means of acceleration (changes in respiration or muscular activity) were demonstrated, but the electroencephalogram demonstrated that psychic activity preceded the acceleration. General investigation indicated that the influencing of the heart rate was chiefly through sympathetic control, but inhibition and augmentation of vagus influence also played a part. Perhaps the most interesting aspect, however, was the development of the link between conscious thought process and autonomic action by the deliberately invoked emotional experience.

[1] *American Heart Journal*, September, 1947.

Abstracts from Medical Literature.

PHYSIOLOGY.

The Effect of Dietary Fat upon Gastric Evacuation.

J. H. ANNEGARS AND A. C. IVY (*The American Journal of Physiology*, September, 1947) state that the effect of fat as it occurs in normally constituted, mixed meals upon gastric evacuation has not been studied previously. This is primarily because available methods for measuring gastric emptying are poorly adapted to the use of such meals. Recently a new method for detecting changes in the volume of the gastric contents has been devised and tested. The method permits the use of meals of any desired composition, and does not interfere with normal gastric activity. In the present study the gastric evacuation of mixed meals containing various amounts of lard or hydrogenated vegetable oil ("Crisco") has been determined in normal subjects. In the experiments thirty normal subjects were given 1500 Calorie test meals of constant weight and volume and containing 53, 77 and 120 grammes of total fat. Of this, 25, 50 and 80 grammes respectively were lard or hydrogenated vegetable oil. Four hours after the test meal the stomach was outlined with a small amount of barium sulphate suspension and an X-ray film was immediately made. Changes in the four-hour volume of the stomach were detected by measurement of the area of the projected gastric shadow. Delayed gastric evacuation attributable to the fat content of the test meal was statistically significant. Twenty-five subjects showed a delay at four hours when the fat content was increased from 6% to 8% and 27 subjects showed delayed gastric evacuation when the fat content was increased from 8% to 14%. No significant differences were found between lard and hydrogenated vegetable oil in their effect on gastric emptying. No gastro-intestinal symptoms followed any test meals with either lard or hydrogenated vegetable oil. The gastric inhibition which occurred when the fat content of the meal was increased was a consistent characteristic for given individuals.

The Effects of Dietary Caloric Restriction on Maturity and Senescence.

Z. B. BALL, R. H. BARNES AND M. B. VISSCHER (*The American Journal of Physiology*, September, 1947) describe the results of experiments to test the effect of long-time caloric restriction upon the reproductive capacity and life span of female A strain mice. Calorie-underfed female mice of the A strain show a fertility index of 0·08 litter per mouse per month during the period to eight months of age, and 0·04 thereafter. When previously underfed females are allowed food *ad libitum* at eight months of age their fertility index rises to 0·65 litter per mouse per month, which approximates the normal for young full-fed females. It is concluded that maturation with respect to reproductive capacity is retarded or postponed when low-Calorie feeding is imposed through a large fraction of the average normal life span. The Calorie underfeeding did not prevent subsequent maturation and normal gestation at an age when full-fed littermate sisters had become nearly sterile from senility, showing a fertility index of only 0·05 litter per mouse per month. In the experiments reported 25% of A strain female mice subjected to lifetime caloric restriction were alive at an age when all of their full-fed littermate sisters had died. Considering the survival beyond 240 days in these experiments, the 50% mortality times for the life-time Calorie-restricted group, the eight-month restricted, subsequently full-fed group, and the full-fed controls were 200, 140 and 125 days respectively. Some factors influencing the significance of these observations have been discussed. The caloric restriction employed in these studies retarded sexual maturation, but did not prevent its occurrence upon subsequent full-feeding and was associated with a tendency toward a longer life span. Caloric restriction for 240 days, with subsequent full-feeding, did not prevent a significant incidence of mammary carcinoma, although it increased the average cancer age above that in comparable non-virgin, fully fed controls.

Infusion Fluids and Burns.

M. D. MCCARTHY AND W. M. PERKINS (*The American Journal of Physiology*, September, 1947) state that a survey of the literature on the treatment of burn shock discloses disagreement among the many investigators regarding the therapeutic efficacy of the various infusion fluids. It appears that, in general, emphasis is shifting from the concept of plasma deficiency and replacement to one of total body fluid distribution as influenced by deficiencies not only in plasma colloid, but also in the extracellular electrolytes, particularly sodium. In their own experiments the major criterion of long-term survival (ten-day period) was used to judge the relative efficacy of the various fluids studied. Homologous plasma, whole blood, erythrocytes (suspended in physiological saline solution) and globin (modified); human serum albumin and human hæmoglobin solution; sodium chloride solution (0·85%); gelatin, oxypolygelatin, sodium chloride solution (1·06%); 6% dextrose solution; and three modifications of Ringer's solution were compared for their effect on the survival of rats subjected to carefully standardized scald burns. The albumin was infused in 4%, 5% and 25% concentration, and the 5% concentration was prepared with three concentrations of sodium chloride in order to evaluate the relative importance of salt and water in albumin solutions. The results indicate that the effectiveness of albumin solution and the other infusion fluids which afforded the highest survival rates could be attributed to the sodium salts and water content. Physiological saline solution, although comparatively ineffective in correcting hæmoconcentration, was found to be as effective as any of the various infusion fluids employed in promoting the survival of rats subjected to scald burns. In general hæmoconcentration was somewhat greater in animals which succumbed than in those which survived. However, the cell:plasma ratio was not of value in the prognosis of individual animals or in comparing infusion fluids. The solutions which provided the most hæmodilution did not always promote higher survival rates, whereas many animals with severe hæmoconcentration in certain of the infusion groups survived. Hæmoconcentration and plasma volume do not appear to be critical factors in the survival of the rats after thermal injury of the type produced.

Influence of Environmental Temperature and Posture on Volume and Composition of Blood.

C. R. SPEALMAN, M. NEWTON AND R. L. POST (*The American Journal of Physiology*, October, 1947) report some experiments on human subjects relating to modification of the volume and composition of the blood induced by chronic exposure (several days) to uncomfortably warm and uncomfortably cold environments and by prolonged maintenance (several hours) of the erect or recumbent posture. The concentrations of plasma protein and hæmoglobin decreased on exposure to heat and increased on exposure to cold. Plasma protein increased progressively in amount during the first few days of exposure to heat and decreased somewhat more rapidly on exposure to cold. These changes probably are permanent adaptations to the environment. A measurable change in amount of circulating hæmoglobin did not occur during an exposure of a week to either environmental extreme. Plasma protein increased in amount when the upright position was maintained and decreased progressively with the subject in the recumbent position. A detectable change in amount of circulating hæmoglobin did not occur during these experiments, which lasted several hours.

The Inhibition of Ventricular Tachycardia.

M. WILBURNE *et alii* (*American Heart Journal*, December, 1947) found that the intravenous injection of 1·3 milligrammes (one-fiftieth of a grain) of atropine sulphate prevented the development of the ventricular tachycardia produced by the rapid intravenous injection of adrenaline in the intact unanæsthetized animal. They state that atropine would thus appear to be of value in preventing ventricular tachycardia and maintaining a sinus tachycardia and that, if so, this would be of particular importance in myocardial infarction in which the evolution of recent ventricular tachycardia to fibrillation is to be averted. They suggest that the recently observed beneficial effects of atropine in lowering mortality in experimental ligation of coronary arteries may be due in large part to its prevention of ventricular tachycardia and terminal ventricular fibrillation.

The Relation of Oxygen in Bone Marrow Blood to Post-Hæmorrhagic Erythropoiesis.

W. C. GRANT AND W. S. ROOT (*The American Journal of Physiology*, October, 1947) report some experimental studies designed to test the hypothesis that anoxia of the bone marrow is a specific requirement for the initiation of erythropoiesis. A technique for the removal of blood samples from the interior of red bone marrow of the unanæsthetized dog is described. Blood so obtained was studied during a period of erythropoietic stimulation induced by a single 30% hæmorrhage. Oxygen, oxygen capacity and hæmatocrit value of bone marrow

and jugular venous blood are decreased after hæmorrhage. A gradual recovery occurs during the following three weeks indicating red blood cell regeneration. Oxygen saturation and oxygen tension, although much decreased immediately after a single hæmorrhage, return to control levels within three to five hours where they remain during the subsequent period of observation. Abolition of the transient decrease in oxygen saturation can be accomplished by repeated removal of small quantities of blood over a period of several days. Under these circumstances the erythropoietic response is the same as that following the single hæmorrhage.

BIOCHEMISTRY.

Hyaluronidase.

J. M. BEILER AND G. J. MARTIN (*The Journal of Biological Chemistry*, December, 1947) found that ascorbic acid and "Dicumarol" inhibited the action of hyaluronidase at concentrations of 0·1 milligramme per millilitre. Of the other compounds tested only rutin showed an inhibitory action, at a concentration of 1·0 milligramme per millilitre. Combining ascorbic acid with the vitamin *P* substances tested caused a marked potentiation of the inhibitory action. This was not true of a combination of "Dicumarol" and hesperidin methyl chalcone. Neither salicylic acid nor salicyluric acid had an inhibitory effect. There was no synergism between salicylic acid and ascorbic acid. Sulphanilamide was without effect.

Sphingomyelin.

S. J. THANNHAUSER AND N. F. BONCODDO (*The Journal of Biological Chemistry*, January, 1948) have described a procedure for the preparation of pure brain sphingomyelin free of hydrolecithin. The fatty acids present in brain sphingomyelin are different from those present in other organs investigated. The component fatty acids of brain sphingomyelin are stearic, nervonic and lignoceric acids, while lignoceric acid and palmitic acid are the component fatty acids of sphingomyelin prepared from spleen and lung. Sphingosine isolated after acid hydrolysis of brain as well as spleen sphingomyelin is a mixture of hydrosphingosine and unsaturated sphingosine.

Acetoacetate and Blood Sugar.

H. C. TIDWELL AND H. E. AXELROD (*The Journal of Biological Chemistry*, January, 1948) found a daily hyperglycæmia in the rat when injected with acetoacetic acid for three consecutive days. The elevation of the blood sugar level apparently was not caused by the ketone body, since the same results were obtained with saline solution or with no injections. Also the blood sugar value, before and after the administration of smaller amounts of the keto acid which had been given daily for three weeks, was normal if blood samples were taken on only one day each week. The glucose tolerance was also unchanged by simultaneous injection of the ketone body. However, the administration of a large amount of acetoacetic acid (140 milligrammes per 100 grammes of body weight) caused hypoglycæmia even after a single injection. The authors' data do not support the view that insulin is inactivated by the keto acid. The hypoglycæmia after administration of large amounts of the acetoacetate is compatible with stimulation of the secreting pancreatic tissue, or a glucose-sparing action associated with decreased glycogenolysis and hence a lowered blood sugar level.

Creatine and Creatinine.

H. D. HOBERMAN *et alii* (*The Journal of Biological Chemistry*, January, 1948) labelled the tissue creatine of a human subject with N^{15} and observed the changes in the amount of creatine deposited in the body before and after the ingestion of a large dose of the non-isotopic compound. It was found that the rate of turnover of endogenous creatine on a diet as free of creatine and creatinine as possible was 1·64% per day. Balance could not be demonstrated between the amount of creatine retained and the amount actually deposited in the depot, or excreted as extra creatinine. To explain these observations it is suggested that the synthesis of endogenous creatine is retarded in the presence of exogenous creatine. The amount of creatinine excreted daily was shown to be directly proportional to the amount of creatine in the body. The uptake of creatine by muscle takes place at a measurable rate. The processes which may be involved in determining this rate are discussed.

Phosphatase and Diabetes.

D. L. DRABKIN AND J. B. MARSH (*The Journal of Biological Chemistry*, December, 1947) have demonstrated increases, which were statistically highly significant, of 23% and 38% respectively in the activities of the acid and alkaline liver phosphatases in rats with well-established alloxan diabetes. Effective insulin therapy applied to rats with well-established alloxan diabetes was found to restore both acid and alkaline liver phosphatase activities to normal levels.

Fœtal Metabolism.

W. H. GOLDWATER AND DE W. STRETTEN (*The Journal of Biological Chemistry*, August, 1947) have shown that the feeding of deuterio fatty acids and deuteriocholesterol to pregnant rats in the latter part of gestation resulted in the appearance of the corresponding deuterio compounds in the fœtuses, proving that these compounds crossed the rat placenta. Enrichment of the body fluids of pregnant rats with deuterium oxide resulted in the rapid incorporation of deuterium into fœtal glycogen, fatty acids, and cholesterol at rates which indicated that these compounds were synthesized in the fœtal organism. The data indicated that, in the rat fœtus approaching the twentieth-day stage of development, half of the fœtal glycogen had arisen by synthesis in twelve to eighteen hours, half of the fœtal fatty acids in about one and a half days, and half of the fœtal cholesterol in about two and a half days. The quantity of glycogen synthesized and deposited in the fœtus per day was found to be approximately equal to the total amount of glycogen present at this stage of development, that is, some 500 milligrammes per 100 grammes of tissue. The synthetic rates in the fœtus were all appreciably higher than the corresponding rates in adult rats. Injection of epinephrine into the pregnant rat caused a drop in the content of fœtal glycogen, this suggesting that epinephrine crossed the placenta and that fœtal glycogen was susceptible to its glycogenolytic stimulus. The injection of insulin into a pregnant rat containing deuterium oxide in its body fluids caused a slight increase in the deuterium incorporated into fœtal glycogen, fatty acids and cholesterol. This is attributed not to any direct action of insulin in the fœtus, but rather to decreased availability of maternal glucose and increased availability of small fragments derived therefrom.

Calcified Tissues.

W. D. ARMSTRONG AND C. P. BARNUM (*The Journal of Biological Chemistry*, January, 1948) have presented methods for the separation of radiocalcium and radiophosphorus in tissues which allow the concurrent use of these isotopes. The uptake and retention of both radiocalcium and radiophosphorus by calcified tissues in the rat stand in the following decreasing order: femur marrow, femur epiphysis, femur diaphysis, incisor tooth dentine, incisor tooth enamel, molar tooth dentine, and molar tooth enamel. Evidence has been presented that under conditions *in vivo* calcified tissues do not all exchange calcium and phosphorus equally.

Liver Enzyme Activity.

L. L. MILLER (*The Journal of Biological Chemistry*, January, 1948) reports that a seven-day fast resulted in a loss of rat liver catalase, alkaline phosphatase, xanthine dehydrogenase and cathepsin activity, which paralleled or exceeded the loss of liver protein. This loss of enzyme activity could not be explained by a loss of prosthetic groups or enzyme activators or by an accumulation of inhibitors and probably represented a loss of enzyme protein *per se*. These findings bear on the relation of liver enzymes to morphological structures, on the metabolism of enzyme proteins during protein catabolic loss, and on the phenomenon of enzyme adaptation in higher mammals.

Amino Acids.

F. FRIEDBERG AND D. M. GREENBERG (*The Journal of Biological Chemistry*, May, 1947) report that insulin, epinephrine, œstrogen, hypophysectomy, thiouracil treatment and thyreoidectomy decrease the blood amino-acid concentration; thyroxine, lipo-adrenal cortex extract and also protein starvation increase it. Adrenalectomy results in hypo-amino-acidæmia, but simultaneously causes a rise in the free amino acid level of muscle. In the kidney, insulin, epinephrine, œstrogen and hypophysectomy produce a significant fall. Thyroxine, lipo-adrenal cortex extract and protein starvation elevate the liver amino-acid level. Thyroxine and lipo-adrenal cortex extract also bring about an increase of the free amino acids in muscle. The amino-acid concentration is highest in kidney and brain and decreases in other tissues in the following order: spleen, liver, testis, muscle. Intravenously administered amino acids are rapidly removed from the blood plasma. They are highly concentrated by liver and muscle, and not at all by brain. The pattern of concentration is not the same for all of the amino acids examined.

British Medical Association News.

REGULATIONS UNDER THE PHARMACEUTICAL BENEFITS ACT, 1947.

THE following extract from the regulations under the *Pharmaceutical Benefits Act*, 1947, is published at the request of the General Secretary of the Federal Council of the British Medical Association in Australia.

4. (1) The Commonwealth Pharmaceutical Formulary shall be in accordance with the Second Schedule to these Regulations.

(2) The addendum to the Commonwealth Pharmaceutical Formulary shall be in accordance with the Third Schedule to these Regulations.

(3) Except in the case of formulæ required to conform to the standards prescribed by the British Pharmacopœia, the British Pharmaceutical Codex or the Australian Pharmaceutical Formulary, and biological products, capsules of amyl nitrite, injections (other than rectal injections), pills, tablets for oral or hypodermic administration, vitamin solutions, a formula in the Commonwealth Pharmaceutical Formulary may be varied—

(*a*) by reducing the quantity of any ingredient;
(*b*) by omitting any ingredient;
(*c*) by adding any drug (other than a drug included in the formula) specified in the Commonwealth Pharmaceutical Formulary in any list of allowable additions applicable to the formula; or
(*d*) by increasing the quantity of any ingredient marked with an asterisk up to the maximum British Pharmacopœia dose for that ingredient.

(4) An ointment base in the Commonwealth Pharmaceutical Formulary may be appropriately varied in a region where the prevailing temperature renders the formula for that base unsuitable.

11. (1) A prescription for the supply of a pharmaceutical benefit shall be in accordance with Form E, and shall indicate by the use of the relevant title included in the Commonwealth Pharmaceutical Formulary or the addendum thereto the name of the pharmaceutical benefit to be supplied.

(2) Subject to this regulation, each prescription shall be written in duplicate on a form supplied by the Commonwealth, and one prescription only shall be written on each prescription form.

(3) A medical practitioner shall not use a prescription form supplied by the Commonwealth otherwise than for the writing of a prescription in accordance with, and for the purposes of, the Act and these Regulations.

Penalty: Fifty pounds.

(4) A medical practitioner shall not write a prescription for the supply of a pharmaceutical benefit unless—

(*a*) he has satisfied himself by personal examination of the person in respect of whom the prescription is written that the pharmaceutical benefit specified in the prescription is necessary for the treatment of that person;
(*b*) that person is suffering from a recurrent, chronic or malignant disease and has previously been under treatment for that disease by the medical practitioner; or
(*c*) it is impracticable for the medical practitioner to examine that person, and adequate particulars of the person's physical condition have been communicated to him.

(5) Where, in a case of emergency, a medical practitioner considers it necessary to write a prescription for the supply of a pharmaceutical benefit, but a prescription form supplied by the Commonwealth is not immediately available for that purpose, he may write a prescription in accordance with Form E on any other suitable paper, and in any such case the provisions of sub-regulation (2) of this regulation shall not apply.

(6) Where a pharmaceutical chemist has supplied a pharmaceutical benefit on presentation of a prescription written by a medical practitioner in accordance with the last preceding sub-regulation, the pharmaceutical chemist—

(*a*) shall, as soon as is practicable, request the medical practitioner to furnish to the pharmaceutical chemist, in lieu of that prescription, a separate prescription, in duplicate, on a form supplied by the Commonwealth in respect of each pharmaceutical benefit included in the first-mentioned prescription; and
(*b*) subject to this regulation, shall not be entitled to receive payment from the Commonwealth in respect of that pharmaceutical benefit unless he has obtained, and produces with his claim for payment, such a separate prescription.

(7) A medical practitioner to whom a request has been made under the last preceding sub-regulation shall, within two days after the making of the request, comply with the request.

Penalty: Fifty pounds.

(8) If the Director-General is satisfied that the pharmaceutical chemist has been unable to obtain from the medical practitioner a prescription in accordance with sub-regulation (6) of this regulation, he shall, upon a claim for payment being made by the pharmaceutical chemist not earlier than one month after he has requested the medical practitioner to furnish such a prescription, direct that production of such a prescription shall not be necessary, and thereupon production of such a prescription shall not be required as a condition of payment for the supply of the pharmaceutical benefit.

(9) A medical practitioner who writes a prescription for a pharmaceutical benefit otherwise than on a form supplied by the Commonwealth shall not write on the prescription a direction that the pharmaceutical benefit be supplied more than once, and if he does write such a direction, the prescription shall be deemed, for the purposes of the Act and these Regulations, to be a prescription for the supply of the pharmaceutical benefit once only.

(10) Subject to these Regulations, a medical practitioner shall not give or send or cause or permit to be given or sent to any person a form in accordance with Form E (whether signed by him or not) unless he has first written on the form, in accordance with these Regulations, a prescription for the supply of a pharmaceutical benefit.

Penalty: Fifty pounds.

24. An approved medical practitioner who has included in a prescription a direction to repeat the prescription shall not himself supply more than once the pharmaceutical benefit specified therein.

25. The Formulary Committee appointed under section 18 of the Act shall advise the Minister on any matter in connexion with the Commonwealth Pharmaceutical Formulary or the addendum to the Commonwealth Pharmaceutical Formulary which the Minister refers to the Committee for consideration or upon which the Committee considers it desirable to offer advice to the Minister.

33. A medical practitioner treating a person in or at an approved public hospital shall not write a prescription in accordance with Form E in respect of that treatment.

34. (1) A person shall not, except in accordance with the Act or these Regulations or with the consent or at the direction of the Director-General, part with possession of, mutilate or destroy—

(*a*) any copy of the Commonwealth Pharmaceutical Formulary, or of the addendum thereto;
(*b*) any copy of any rules; or
(*c*) any list or form,

supplied to him by or on behalf of the Commonwealth under or for the purposes of the Act or these Regulations.

(2) The Director-General may, by notice in writing served on a person, require that person to deliver up to the Director-General, or to a person specified in the direction, within a time specified in the direction, any document or other paper specified in the last preceding sub-regulation which is in the possession of the person on whom the notice is served, and that person shall comply with the direction.

Penalty: Fifty pounds.

35. A notice or other document under the Act or these Regulations may be served by post on the person or authority to whom it is addressed at any premises in respect of which he or it is approved for the purposes of the Act or at his or its last known place of business or practice.

Reg. 11. Form E.

COMMONWEALTH OF AUSTRALIA.

Pharmaceutical Benefits Act, 1947.

Prescription Form.

NAME.
ADDRESS.
DATE.

N.B.—One Formulary item only to be written on this Form, and title of the pharmaceutical benefits, as shown in the Commonwealth Pharmaceutical Formulary, to be indicated.

℞ (Signature of medical practitioner.)
(Address.)

MEDICO-POLITICAL.

A MEETING of the Victorian Branch of the British Medical Association was held at the Medical Society Hall, Albert Street, East Melbourne, on April 7, 1948, Dr. F. Kingsley Norris, C.B.E., D.S.O., the President, in the chair.

Financial Statements.

Dr. J. A. Cahill, the Honorary Treasurer, presented the statement of receipts and expenditure for the year ended December 31, 1947. The statements were received and adopted. The statements are published herewith. In presenting the statements Dr. Cahill spoke in the following terms.

A rule of the Branch imposes on the Honorary Treasurer the obligation of presenting to the February meeting of the Branch an annual balance sheet.

Unfortunately this year it was not practicable to discharge that duty in February or even in March, for reasons which I explained at the February Branch meeting. The whole of the time available at that meeting was occupied in explaining the necessity for increasing members' subscriptions and in presenting and passing the necessary resolutions. Owing to the centenary celebrations at the Royal Melbourne Hospital there was no March meeting of the Branch.

I shall therefore tonight at this earliest opportunity discharge in part my obligation and present a statement of receipts and expenditure. Next year it will be discharged

BRITISH MEDICAL ASSOCIATION—VICTORIAN BRANCH AND THE MEDICAL SOCIETY OF VICTORIA.

Combined Statement of Receipts and Expenditure for Year Ended December 31, 1947.

RECEIPTS.	£ s. d.	£ s. d.
To Balance at December 31, 1946—		
National Bank of Australasia, Limited—Medical Society of Victoria	2,241 4 10	
State Savings Bank of Victoria	504 9 4	
Commonwealth Loans	500 0 0	
Cash in Hand	17 0 0	
	3,262 14 2	
Less National Bank of Australasia, Limited, Overdraft, British Medical Association	2,197 14 8	
		1,064 19 6
„ Subscriptions		11,880 1 8
„ British Medical Insurance Company—Grant		300 0 0
„ Sales of Journals		2 10 0
„ Sales of List of Members		16 0 6
„ Interest on Investments		26 5 10
„ Contributions and Donations to Funds—		
Victorian Medical Benevolent Association	351 2 10	
Federal Medical War Relief Fund	104 7 0	
Food for British Doctors	694 18 0	
British Medical Association Legacy	999 8 6	
Crawford Mollison Prize	253 3 0	
		2,402 19 4
„ Debentures—		
British Medical Insurance Company	175 0 0	
Debenture Redemption Account	200 0 0	
		375 0 0
		£16,067 16 10

EXPENDITURE.	£ s. d.	£ s. d.
By British Medical Association, London		2,509 4 11
„ THE MEDICAL JOURNAL OF AUSTRALIA		1,047 16 10
„ Federal Council of the British Medical Association in Australia—Capitation Fees		958 10 0
„ Rebates to Subdivisions		35 14 0
„ Medical Society of Victoria—Organization Fund		1,461 16 0
„ Medical Society of Victoria—Debenture Redemption Account		164 8 4
„ Library Books and Journals *et cetera*	398 7 0	
Less Contribution from British Medical Insurance Company	120 0 0	
		278 7 0
„ Salaries—Medical Secretary	1,300 0 0	
„ Office and Library *et cetera*	1,886 0 5	
		3,186 0 5
„ Life Assurance Premium		162 17 6
„ Fund Disbursements—		
Victorian Medical Benevolent Association	351 2 10	
Federal Medical War Relief	104 7 0	
Food for British Doctors	167 9 6	
British Medical Association Legacy—Commonwealth Loan	50 0 0	
		672 19 4
„ Debentures—Australasian Medical Publishing Company, Limited		896 18 2
„ Debenture—		
Interest		183 15 0
Principal		375 0 0
„ Audit Fees		22 1 0
„ Advertising		2 2 8
„ Postages		345 18 0
„ Printing and Stationery		322 17 3
„ Sundry Expenses		150 2 6
„ Bank Charges and Exchange		3 4 11
„ Telephones		78 8 2
„ Lighting and Heating		64 17 3
„ Lanternist		2 0 0
„ Gas Radiator Heating		7 12 11
„ Office Furniture and Equipment		193 8 2
„ Rates, Taxes and Insurance		73 19 8
„ Repairs		18 13 3
„ Legal Expenses		77 12 4
„ Travelling Expenses		1 17 2
„ Entertainment		45 3 11
„ Donations		2 12 0
„ Australasian Medical Publishing Company, Limited—List of Members		16 0 6
„ Balance at December 31, 1947—		
National Bank of Australasia, Limited—Medical Society of Victoria	4,269 0 10	
State Savings Bank of Victoria	514 10 2	
Commonwealth Loans	500 0 0	
Cash in Hand	19 0 0	
	5,302 11 0	
Less National Bank of Australasia, Limited, Overdraft, British Medical Association	2,596 13 4	
		2,705 17 8
		£16,067 16 10

We report that we have compared the above Statement with the ordinary audited Statements of Receipts and Expenditure of the British Medical Association—Victorian Branch Account and the Medical Society of Victoria Account and found them to be in accordance therewith.

(Signed) J. V. M. WOOD AND COMPANY,
Chartered Accountants (Aust.).

Melbourne, January 30, 1948.

(Signed) J. A. CAHILL,
Honorary Treasurer, January 30, 1948.

in full as an alteration has been made in the accounting system that will permit of the presentation of a profit and loss statement and a balance sheet. These I consider should be made available to members.

I shall now briefly refer to the statement—copies of which have been distributed to you—covering the financial transactions of the Branch for the year ended December 31, 1947.

It will be seen that the year opened with a credit balance of £1,064 19s. 6d. and closed with a credit balance of £2,705 17s. 8d. These figures, however, do not reveal the correct position. To arrive at what the position actually is, or was, a little analysis is necessary. On the "Receipts" side will be seen included under the heading "Contributions and Donations to Funds": Food for British Doctors £694 18s., B.M.A. Legacy £999 8s. 6d., Crawford Mollison Prize, £253 3s., with contra entries only for Food for British Doctors at £167 9s. 6d. and B.M.A. Legacy of £50 on the expenditure side. In the National Bank to the credit of the Medical Society of Victoria Account there was therefore a sum of £1,730 that was contributed by members for specific purposes which had no relation whatever to the Branch as such. These amounts were so deposited by way of convenience and have since been transferred to their respective Trust Accounts to be applied to the purposes for which they were donated. The collections for those funds, however, being where they were at December 31, 1947, had the effect of increasing the credit balance by £1,730. That amount has therefore to be deducted to arrive at the correct position. The credit balance of the combined British Medical Association and Medical Society of Victoria accounts at the end of the year was consequently £975 17s. 8d.—that is, £2,705 17s. 8d. less £1,730.

You will now realize that there was another loss on the year's transactions in spite of a subscription revenue of £11,880 1s. 8d., a record for the Branch, and generous subventions amounting to £420 from the British Medical Insurance Company. Members are now familiar with the steps taken to provide against these recurrent losses and to meet the increased expenditure involved in the increased Federal capitation fee to 15s. and other increases in expenditure that had to be budgeted for if a recession of the Branch's activities had to be avoided when there was a vital demand for greater activity, not only within the State but throughout the Federal sphere. There is no need for me to be more specific in that regard. When putting the case to the Branch meeting in February for increasing the subscriptions I dealt in detail with these matters.

Other items on the "Receipts" side, of which I think some special mention is necessary, show that the generosity of members continues unabated notwithstanding heavy taxation and high cost of running practices. Contributions to the Medical Benevolent Association totalled £351 2s. 10d., to the Federal War Relief Fund £104 7s., and to the other funds I have already mentioned £1,947 9s. 6d., making a substantial total of £2,402 19s. 4d.

Items on the "Expenditure" side, with perhaps one exception, are self-explanatory. The item upon which I think an explanation is called for is "Debentures—Australasian Medical Publishing Company, Limited, £896 18s. 2d.". Briefly, the explanation of that item is this. A proposal has been accepted by the Branch under which, of the £1 *per annum per capita* payment to the Australasian Medical Publishing Company, Limited, for the journal, 12s. 6d. shall be regarded as subscription for the journal and 7s. 6d. shall be hypothecated for the purchase of debentures to be issued in the name of the Medical Society of Victoria bearing interest at 3½%. In other words, whereas formerly the whole of the £1 *per capita* payment for the journal was regarded as a straight out subscription, in future 7s. 6d. of the amount will be a loan to the Australasian Medical Publishing Company, Limited, from which the Branch will receive a small cumulative interest income. The company in turn will apply the money so loaned for necessary extension of buildings, plant and equipment.

I have already referred to the generosity of the British Medical Insurance Company. I would reiterate that this generosity is made possible only through the profit earned by the company. To make a profit, the company must write business. Therefore, in your own interests, I would urge all members to place with the British Medical Insurance Company whatever insurance business they may be able to direct.

I would like to also direct your attention to the British Medical Agency in which you have a direct interest and which relies on your support. You will remember how, three years ago, substantial donations from the British Medical Insurance Company and the British Medical Agency made possible the purchase by the Medical Society of Victoria of the property known as 384-6, Albert Street, East Melbourne, the site on which it is hoped to erect a building worthy of the Association.

I now formally move that the statement of receipts and expenditure for the year ended December 31, 1947, be received and adopted.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at the Children's Hospital, Carlton, Melbourne, on November 19, 1947. The meeting took the form of a series of demonstrations by members of the honorary medical staff of the hospital and was combined with the monthly meeting of the Melbourne Pædiatric Society.

The Cœliac Syndrome.

DR. J. W. GRIEVE and DR. DAVID PITT demonstrated five patients illustrating the cœliac syndrome. Two were classical examples of subjects of cœliac disease, one an infant and the other a girl, aged twelve years, who had been recognized as suffering from cœliac disease in 1939 and who had made excellent progress, at the present time revealing a protuberant abdomen only. The other three were suffering from fibrocystic disease of the pancreas recognized after they were six months of age; they had progressed relatively satisfactorily on a diet with high protein, low fat and low starch content *plus* extra protein and pancreatin. One of the patients had quite dramatically sustained a weight gain after the introduction of pancreatin over twelve months previously. Dr. Pitt illustrated the diagnosis, management, investigation and pathology of these patients with appropriate charts, diagrams, pathological specimens and sections. The object of showing the patients was to indicate that a subgroup of the cœliac syndrome consisted of fibrocystic disease, which could very often be recognized in early infancy and was frequently associated with severe pulmonary infections leading to bronchial occlusion and bronchiectasis. The pathological investigation of some of the older children had revealed widespread fibrosis in liver, spleen and lungs as well as in the pancreas. Recently a section of a salivary gland of one child had revealed the same characteristic changes. The progress of one of the patients with fibrocystic disease of the pancreas after the introduction of pancreatin suggested that therapy with that drug, *plus* the parenteral administration of vitamin preparations, might be helpful in a certain proportion of those unfortunate patients.

Modification of Cow's Milk for Infant Feeding.

From a number of prepared specimens DR. ROBERT SOUTHBY demonstrated the various modifications of cow's milk to make it suitable for use in the feeding of infants. The samples were designed to give an in-vitro visual impression of what actually happened *in vivo* in the stomach of the infant. For that purpose tubes containing the various types of feeding were treated with hydrochloric acid and rennin and kept at body temperature for thirty minutes; the various samples were then sealed in separate tubes for demonstrating purposes.

The first series showed the tough casein curds vomited by the infant who could not digest whole cow's milk, and the typical bean-shaped "casein curds" passed in the stools by the same infant. The next group comprised samples of colostrum, breast milk and cow's milk to illustrate the main gross differences between those forms of feeding. A further group showed the appearance of whole cow's milk, boiled milk, milk and water of equal parts, and citrated milk; the curd varied from large tough masses in the whole milk to lighter and less bulky curds in the diluted or boiled milk, and finally practically no curd whatever in the citrated modification. A final series showed dried milk, condensed milk, lactone syrup milk, and "Bengerized" milk, all of which presented little or no curd formation. It was plainly evident that the one point in common with all the modified feedings was that the tough casein curd had been changed to a greater or less degree so as to provide a much more easily managed food for the infant stomach to deal with.

To amplify the matter further Dr. Southby also demonstrated the Hill Curd Tension Test in which the actual tension in the curd was estimated by the number of grammes weight necessary to draw the curd tension knife through a specified quantity of the particular curd undergoing the test. By means of the test it was easily shown that, whereas the curd tension figure for breast milk was practically nil, that for cow's milk was in the region of 200 grammes. The

practical value of the test, according to Dr. Southby, was that it allowed of the selection of soft curd milk for infant feeding and of hard curd milk preferably for cheese making. He said that that was quite possible on a practical scale since the curd tension figure for any one cow was more or less constant and independent of the time of day or season of the year in which the test was carried out.

Intracranial Hæmorrhage of the Newborn.

Dr. Southby next showed a series of babies who had suffered from intracranial hæmorrhage in the neonatal period, and he emphasized that the condition was one of the most urgent emergencies in pædiatrics. He said that the newborn baby who was disinclined to suckle at the breast readily and naturally, who was listless and apathetic and perhaps subject to pale or cyanotic turns, especially if there was any suspicion of restlessness, twitching, or generalized convulsions, should be suspected of having some grave intracranial lesion, most likely one of hæmorrhage. When that was suspected lumbar puncture or a subdural tap on one or both sides of the skull was indicated in order to determine the diagnosis. At that juncture Dr. Southby showed four specimens of cerebro-spinal fluid to indicate the progressive changes in its macroscopic appearance as the condition subsided: (*a*) bright red frankly blood-stained fluid; (*b*) dirty rusty brown fluid; (*c*) yellow straw-coloured fluid; (*d*) crystal clear fluid. He went on to say that very occasionally the infant might be fortunate enough to survive the lesion with lumbar punctures alone and to come through without any untoward sequelæ. More often a subdural tap would need to be performed, preferably on both sides, and perhaps to be repeated daily for several days until the condition was under control; after that operation for evacuation of clot and debris and irrigation of the site of the hæmatoma might be carried out. Localization of the collection of blood was frequently made more accurately by the aid of encephalography and ventriculography.

Dr. Southby acknowledged the very great help and cooperation of Dr. E. Graeme Robertson and Dr. R. S. Hooper in dealing with the babies and stated that the excellent results obtained in a number of instances were entirely due to efficient teamwork on the part of those specialists. One infant's condition had been diagnosed on the third day after a convulsion and the appearance of some localizing signs, and the site of the collection was confirmed by encephalogram. That baby had been operated upon by Dr. Hooper, had made an excellent recovery and was now progressing remarkably well, although still somewhat slow in getting onto his feet. A second infant was one of twins, the other being quite normal. The affected twin was difficult to resuscitate after birth, would not feed at all well and was noticed to be disinclined to use the left arm; later when endeavouring to stand the child was noticed to be weak and spastic in the left leg. An encephalogram aroused the suspicion of the presence of a subdural cyst following an old hæmatoma. That baby also was operated upon by Dr. Hooper and had since improved very considerably. The other infants had improved rapidly with frequent lumbar punctures alone and appeared to be free of sequelæ, although it was too early to be definite about the ultimate prognosis.

Dr. Southby stressed the importance of early diagnosis of the condition in the newborn; it was more frequent than was generally realized and offered more prospect of benefit for the baby the earlier an attempt was made to aspirate or to evacuate the extravasated blood.

Finally Dr. Southby summarized the main groups of causes of intracranial hæmorrhage in the newborn as follows: (*a*) premature stillbirth; (*b*) long or difficult instrumental delivery; (*c*) precipitate delivery of a *multipara* immediately after the rupture of the membranes; (*d*) a manifestation of hæmorrhagic disease of the newborn, occurring as it sometimes does in association with hæmatemesis or melena of the newborn; (*e*) an unexpected occurrence in the occasional normal non-instrumental delivery.

Hæmatemesis in Children.

Dr. Southby's last patients were two girls, aged respectively nine and three years, who had come to the hospital with a history of hæmatemesis of sudden onset; both appeared extremely ill on admission. The younger of the two proved on investigation to be suffering from *Hæmophilus influenzæ* meningitis and was responding as well as could be expected at the present juncture to treatment with specific serum, streptomycin, penicillin and sulphadiazine. Dr. Southby felt that the child was of interest in showing the frequency with which acute meningitis, especially in infants and young children, could be heralded by a gastro-intestinal type of onset; in any such child, acutely ill particularly with marked pallor, the possibility of meningitis must be ever in mind and there should be no hesitation in performing a lumbar puncture if no other exciting cause of the severe symptoms could be located.

Dr. Southby said that the older child had been under observation for a possible tuberculous chest lesion. She had suddenly had what was thought to be a severe hæmoptysis, but was found to have a hæmoglobin value of 50% and a total red cell count of just under two million; further observation revealed that the condition was really one of hæmatemesis. The investigations all revealed nothing abnormal, and there was no enlargement of either the liver or the spleen. Dr. Southby considered that the child's condition would most likely prove to be one of the liver-spleen types of trouble, although there was no visceral enlargement at the time of presentation.

(*To be continued.*)

Correspondence.

A JOURNAL OF ELECTROENCEPHALOGRAPHY.

SIR: At the International Electroencephalography Meeting held in London in July, 1947, it was proposed that an *International Journal of Electroencephalography and Clinical Neuro-Physiology* should be founded with Dr. H. H. Jasper (Montreal) as editor-in-chief and with Dr. W. Grey Walter (Bristol) as co-editor and with special responsibility for Europe.

Dr. Walter has asked if it would be possible to estimate and perhaps obtain a list of subscribers in Australia to this projected journal.

Dr. Robert S. Schwab, of the Massachusetts General Hospital, Boston 14, Massachusetts, United States of America, is handling the correspondence for the Pacific and Far East, and if any members of the medical or teaching profession who are interested could contact him in this matter it would be most helpful and accelerate the publication of this very necessary journal.

Alternatively they could write to me and I will be pleased to forward to Dr. Schwab the information they make available.

Yours, etc.,
GEOFFREY TRAHAIR.

Royal Prince Alfred Hospital,
Camperdown,
New South Wales.
April 23, 1948.

PERCUSSION OF THE SPINE.

SIR: On a recent examination of a patient I have found a distinct alteration in the percussion sound of his spine. This finding attracted my attention as I have been always keen on this subject. When quite a young assistant I had the opportunity to watch the investigations on the percussion of the spine by my first clinical chief, Professor Fr. Korányi. Korányi claimed the priority to the *"trigonum paravertebrale"*, the downwards broadening triangular dulness on the lower part of the dorsal vertebral column and on the opposite side of the thorax in case of a pleural effusion. The Italian and English textbooks call it the triangle of Grocco (who described it in 1902); the German literature terms it mostly as the triangle of Rauchfuss (1903).

This paravertebral triangle spurred Korányi (who described it in 1897) in his last years to further investigations about the percussion of the vertebral column. This research was continued by his famous son, A. Korányi, the creator of the modern concept of the insufficiency of the kidney; he was the first to use and recommend the determination of the freezing point of the blood serum as a routine clinical investigation. He extensively examined the alteration of the location and shape of kidney dulness in the various diseases of this organ.

In my diagnostic lectures at the University of Budapest I taught for thirty years that on the first three or four dorsal vertebræ the percussion sound is slightly dull, from the fourth to the twelfth the sound is loud, then follows the marked dulness of the lumbar vertebræ. Tumour in the mediastinum, aneurysm of the aorta, especially of the

descending aorta, produce a marked dulness on the fifth to seventh dorsal vertebræ, occasionally even a little further downwards. Such alteration may sometimes be caused by a big intrathoracic goitre also, as I had occasion to demonstrate in a case some months ago.

Changes in the percussion sound of the spine can be produced by several other diseases too, for example, by a big mass of enlarged peribronchial lymph glands. Fluids in a very dilated œsophagus give a well-marked dulness on the lower half of the dorsal spine. I remember having seen many years ago a case of a big mediastinal abscess caused by perforation of the œsophagus by a swallowed bone. It gave a distinct dulness on the dorsal spine. I also recollect a case of post-operative mediastinal emphysema where the percussion sound on the third to sixth dorsal vertebræ seemed quite abnormally loud and sonorous.

I mention all this because in the up-to-date English and American treatises on medical diagnosis and internal medicine I hardly found any notice about this subject. It is mentioned in the books of Greene and of Cabot and Adams; in many others it is not referred to at all. As I personally feel that the percussion of the spine may occasionally offer valuable information, I feel that I should not suppress my opinion that it would be a pity to neglect it completely. Could somebody give me his own impression? I would be very interested and obliged indeed if anyone were to inform me about his experiences on this subject. I firmly believe that it is not futile to deal with this question.

Yours, etc.,

CHARLES ENGEL, M.D.

22, Ocean Avenue,
Edgecliff,
New South Wales.
April 26, 1948.

Bibliography.

A. Korányi: *"Vorlesungen über funktionelle Pathologie und Therapie der Nierenkrankheiten"*, 1929, page 22.

C. Engel: "Percussion", in *"Belorvosi diagnosztika"*, by E. Jendrassik, 1921, page 62.

C. Greene: "Medical Diagnosis", 1922, page 297.

R. C. Cabot and F. D. Adams: "Physical Diagnosis", 1938, Figure 116.

MEDICAL MISSIONS: AN APPEAL.

SIR: The work of the Anglican Mission in New Guinea stretches along the North Coast from East Cape to the boundary line separating Papua from the old mainland Mandated Territory. Not only are its stations dotted along the whole length of that rough coast, but they also go far inland among the tribes that inhabit the Owen Stanley Ranges.

For the last ten or more years there has been no doctor located on that long coastline between Samarai and Lae. During the war military and air force doctors rendered splendid service both to the natives and also to the isolated missionaries who remained at their posts during the Japanese onslaught. Since then a small band of triple certificated missionary nursing sisters has scattered its members far and wide in giving a most valiant ministry to white and brown folk alike, but there has been no doctor. In a short three months' sojourn on the coast in 1946 I became greatly indebted to one of them during a sharp attack of dysentery and the experience made me realize a little of what it must mean to permanent workers and residents to be without the ministry of a qualified doctor.

Dr. Blanche Biggs, a Tasmanian born and a University of Melbourne graduate, has recently offered her services as a medical missionary. After taking one or two specialized courses she expects to be ready to start at the beginning of July. There lies before Dr. Biggs the work of establishing and organizing one (and possibly two) base hospital, and many medical outposts in country where roads are for the most part an unknown luxury. A large part of her travelling will also be done in a launch up and down the coast. Her isolation from medical fellowship will be very complete.

For a man this would be a formidable task to contemplate. That a woman should be undertaking it has made most of us feel that in sheer respect for her grit we must do all that is in our power to give her the finest possible backing. We aim to provide her with the completest medical equipment possible, and thus to enable her to atempt to do a scientific job of the highest value. We think this is the least we can do, and we estimate that the outlay will be at least £2000.

It has occurred to us that the medical world of Australia is likely to be deeply interested in this courageous enterprise. Therefore, with the commendation of some members of the medical profession, we are taking the liberty of inviting the cooperation and help of Dr. Biggs's fellow medical men and women throughout the country.

Yours, etc.,

G. H. CRANSWICK, Bishop,
Chairman, the Australian Board of Missions.

14, Spring Street,
Sydney,
April 28, 1948.

TREATMENT OF THE EYE IN THE CONSULTING ROOM.

SIR: Disagreement must be expressed with two statements of your reviewer of "Office Treatment of the Eye", April 17, 1948.

He apparently does not agree that active measures should be instituted if detachment of the chorioid persists for a month after a fistulizing operation. Fortunately, the condition is a rare one, so rare that Stallard does not give any reference to it in his recent book, nor does Spaeth, who includes every possible shade of opinion in his treatise. The latter does mention a persisting hypotony without reference to the cause. After a trephine some years ago I had the misfortune to encounter the condition. The chorioid became detached and remained so with disastrous results. Some time later a senior colleague trephined the other eye with no better fortune. On two occasions since after trephining an eye I have had a detachment of the chorioid which lasted a month. Fearing that the hypotony might become permanent, I used Juler's technique, and transplanted a conjunctival graft in each. The chorioid went back into position, and the tension rose to normal. The procedure is quite harmless and not difficult. As the outlook with persistent detachment of the chorioid is so grave, I feel that it should be undertaken in every case in which the detachment has lasted a month.

The statement that "the presence of pannus in trachoma is not usually regarded as a contra-indication to the use of copper-sulphate, as is stated by the author", must be a misprint. He is a brave soul who would not only diagnose trachoma in the absence of pannus, but also institute treatment with copper sulphate.

Yours, etc.,

KEVIN O'DAY.

33, Collins Street,
Melbourne, C.1,
April 23, 1948.

MEDICAL EDUCATION.

SIR: The leading articles on medical education in THE MEDICAL JOURNAL OF AUSTRALIA of January 10 and May 1, 1948, and the short report of the discussion at the recent meeting of the Federal Council call for some comment.

It is not to be wondered at that members of the Federal Council are not eager to commence work on this problem, for it is a time-consuming and difficult one. The members of the Council are no doubt all busy people who realize the magnitude of the suggested undertaking.

There was published in 1946 by the University Publications Committee of the University of Queensland a "Critical Review of Undergraduate Medical Training". The preparation of this review occupied a period of two years and entailed much work on the part of the committee responsible for its production. The booklet was widely distributed both in Australia and abroad.

It should be stated, for the information of those who are interested, that the President of the British Medical Association in Queensland is an *ex officio* member of the Faculty of Medicine. As there are usually a number of past presidents on the Council of the British Medical Association, this body should be well informed on undergraduate medical education in Queensland. Furthermore, other members of the Council are also members of the several boards of studies associated with the Faculty.

All this apart, however, if the Federal Council is able to prepare a report on the subject it would no doubt be welcomed by the medical faculties in the various Australian universities.

In recent years many excellent reports have been received from overseas, including some from Great Britain and the United States of America.

In all the consideration that has been given to the question of medical education since the first world war, no definite decisions have been arrived at on several important aspects of the matter, namely: (i) the restriction of the content of the undergraduate medical course within reasonable compass (for example, the extent of our knowledge of each subject of the course has increased enormously, and as was pointed out in England many years ago, people responsible for the teaching of the subjects are all too eager to reduce the content of those of their colleagues rather than their own); (ii) the problem of improvement of methods of teaching—particularly in the pre-clinical work; (iii) the problem of securing increased financial support for medical education, in order to improve existing facilities—particularly facilities for hospital training, including the provision of adequate hospital residential accommodation for students.

May I say in conclusion that these opinions are made in a purely personal capacity, and that anyone may check the facts in published documents.

Yours, etc.,
E. S. MEYERS.

The University of Queensland Medical School,
Herston,
Brisbane.
May 4, 1948.

THE PHARMACOLOGY OF JUNCTIONAL TRANSMISSION.

SIR: I have studied the article by Professor F. H. Shaw in the journal of April 10, 1948, and find it rather difficult to understand his arguments about nicotine and potassium in the section headed "Sympathetic Ganglia".

He states:

> The main question is whether the action of potassium is prior to that of acetyl choline. If the ganglion is perfused with curarine, acetyl choline is liberated, but is unable to act, while potassium can still initiate a discharge. The action of potassium is abolished by nicotine, but the salt can still cause an antidromic discharge along the preganglionic fibre; thus the action of nicotine is located at the postganglionic cell.

I should like to know what Professor Shaw means by the term "postganglionic cell". Surely this should read "ganglionic cell" or "surface of the ganglionic cell".

Professor Shaw continues:

> It has been suggested that potassium is the transmitter and that it activates the preganglionic elements.

Once again if my understanding of this subject is at all clear the term "preganglionic elements" should read "ganglionic cell" or "ganglionic elements".

Yours, etc.,
P. R. BULL, M.B., B.S.

Austin Hospital for Cancer and Chronic Diseases,
Heidelberg, N.22,
Victoria.
April 30, 1948.

PNEUMONOKONIOSIS IN NEW SOUTH WALES COAL MINERS.

SIR: I do not wish to enter any controversy as to the relative merits of the "old" and the "new" pneumonokoniosis boards—a medico-political subject perhaps better left out of scientific matters. However, Dr. I. A. D. Graham's letter in the journal of April 24, 1948, prompts observations on two aspects of dust disease, applicable not only to coal miners, but to all dusty occupations in which there is a hazard—real or alleged. (I use "pneumonokoniosis" in its general sense to include all pulmonary disease due to the inhalation of dust.)

Dust "phobia" is a very real entity in many of the dusty trades, and in some "nervous" individuals causes almost as much anxiety, industrial disquiet and suffering as the disease itself. This is in no wise meant to belittle the presence of pneumonokoniosis in industry. We do not deny the dangers of cancer by admitting that cancer "phobia" exists. The prevalence and strength of this dust phobia in any industry or community depends in the main on (*a*) the number of cases diagnosed as pneumonokoniosis; (*b*) the amount of publicity—true or false—given to the dust hazards; (*c*) the isolation of the community or industry. Incorrect diagnoses can have almost as great an effect as the presence of true cases dying, as Dr. Graham aptly puts it, "the interminable pulmonary death of pneumonokoniosis". The latter is certainly a sight which strikes fear into the most unimaginative of their fellow workers. In regard to publicity, in some trades there has, over the last decade, been a great spate of it—some of it exaggerated or containing half-truths. When a stage is reached that every symptom referable to the respiratory or cardio-vascular systems is fearfully ascribed to "dust", then surely there must be an increase in claimants appearing before the relevant board and a corresponding increase in the percentage of claims rejected. It is our duty not only to clean up the real hazards, but to reassure these unhappy, anxious men working in situations where the hazard is purely imaginary.

The question in each case would seem, firstly, to determine the amount of disability, if any, and, secondly, its cause. On these points I gather that Dr. Graham would like some objective facts derived from measurement rather than a mass of clinical opinion. That radiological appearances do not necessarily have—in any one individual patient—much bearing on the amount of disability present has been accepted for quite a time. Rubin, therefore, is hardly preaching a new doctrine. In spite of a bewildering array of methods designed to assess disability—including various forms of exercise tolerance tests, spirometry, bicycle ergometry, and even calculations based on changes in blood chemistry—we still are without a foolproof field method which can completely eliminate effects due to the mental state of the patient. It is a subject which constantly exercises the minds of industrial physiologists. Many cases are quite evident, but it is the borderline cases which cause all the forensic affrays and all the bitterness and heartburning. In the doubtful cases we probably end up by relying on clinical impressions as much as on anything else.

Having once decided the degree of disability, the question of cause has to be determined. In most cases the subjects are middle-aged. Fortunately, in many the cause is not a matter of dispute. But there is a substantial group in which the cause may lie between early pneumonokoniosis, moderate degenerative changes in the cardio-vascular system or decreasing physical fitness for hard work due to "ordinary" aging processes—or a subtle blend of all three. Occasionally more unusual and serious causes are found. In other words, the answer becomes inevitably a matter of clinical opinion, and the more experienced the members of any board are, the more likely they are to be right. At all events, there is very little hope of obtaining cut-and-dried facts rather than opinions. The law, and possibly the public, require more clear-cut decisions than we can give them. This was pointed out years ago by the late Charles Badham, who is also reported to have made the most apt summary of the matter to the effect that, in doubtful cases, medical men can, in the light of present knowledge, give "only rough justice".

Dr. Graham's constructive suggestion that the addition of a legal member or members might help to clarify decisions is a moot point, when it is considered that the chairman of the board, the findings of which caused all the controversy, qualified in law some ten years before he qualified in medicine.

Yours, etc.,
DOUGLAS GORDON.
Medical Officer in Industrial Hygiene.

Department of Public Health, Queensland,
Brisbane,
May 7, 1948.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that the following films will be shown at the film evening to be held on Wednesday, May 26, 1948, at 2 p.m. at the Stawell Memorial Hall, Royal Australasian College of Physicians, 145, Macquarie Street, Sydney: "Progressive Muscular Atrophies, Dystrophies and Allied Conditions", "Friedreich's Hereditary Ataxia and Little's Disease", "Chorea", documentary film. All members of the medical profession are invited to attend. Further

inquiries should be made from the Secretary of the Post-Graduate Committee in Medicine, 131, Macquarie Street, Sydney (telephone: B 4606, BW 7483).

Obituary.

EDNA NELSON.

THE death of Dr. Edna Nelson has deprived the community of one of its most able women; it came at a time when she herself was reaping the reward of the years of unremitting care and devotion to her home and her professional work.

After graduation from the University of Sydney with second class honours in 1920 Edna Nelson, or Edna Smith as she was before her marriage, was appointed to the resident medical staff at Sydney Hospital. She also won the Dagmar Berne Prize which is awarded to the most successful woman graduate in her final year. After several years of private practice in Sydney she married Dr. W. T. Nelson and accompanied him to Kalgoorlie. She returned with her husband and young family to Melbourne, where she carried on her work and was appointed to the staff of the Queen Victoria Memorial Hospital where she first took a prominent place in the specialty of veneral diseases in women and diseases of the skin.

On her return to Sydney in 1936 she was appointed director of the venereal diseases clinic at the Rachel Forster Hospital for Women and Children and also assistant physician for the diseases of the skin. In 1939 she went abroad to further her studies in the latter specialty. Since that time she relinquished her association with the venereal diseases clinic, but retained her appointment as honorary physician for diseases of the skin at the Rachel Forster Hospital. She also held an appointment in the dermatological clinic at the Royal Prince Alfred Hospital.

Both in her hospital and private practice Edna Nelson not only inspired confidence and affection in her patients, but was an inspiration to her colleagues who knew and worked with her. The same enthusiasm and whole-hearted interest were given to the public organizations with which she was connected. Her standard of values was high and her integrity in all matters of public interest was of quality only too rare in these days of opportunism. As President of the Business and Professional Women's Club she spared no efforts to advance those ideals on which the club was founded. She was active in furthering women's interests and in trying to secure for women in general the same emoluments and privileges as those enjoyed by men.

Edna Nelson stood high in the estimation of her friends and fellow workers, and will be long remembered for her character and personal qualities. She is survived by her husband and four sons, three of whom are medical students.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Himmelhoch, Albert, provisional registration, 1947 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Books Received.

"Paravertebral Block in Diagnosis, Prognosis and Therapy: Minor Sympathetic Surgery", by Felix Mandl, M.D., F.I.C.S., translated by Gertrude Kallner, M.D., with a foreword by Max Thorek, M.D., F.I.C.S.; 1947. London: William Heinemann (Medical Books), Limited. 9" x 6", pp. 346, with illustrations. Price: 32s.

"Communicable Diseases", by Franklin H. Top, A.B., M.D., M.P.H., F.A.C.P., and Collaborators; Second Edition; 1947. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 8¼" x 5¾", pp. 992, with many illustrations, most of them coloured. Price: 71s.

"Synopsis of Neuropsychiatry", by Lowell S. Selling, M.D., Ph.D., Dr.P.H., F.A.C.P.; Second Edition; 1947. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 7¾" x 4¾", pp. 562, with illustrations. Price: 49s.

"Disputed Paternity Proceedings", by Sidney B. Schatkin; Second Edition; 1947. New York: Matthew Bender and Company Incorporated. 9" x 6", pp. 624.

"Textbook for Almoners", by Dorothy Manchée, with a foreword by Sir Alfred B. Howitt, C.V.O., M.D.; 1947. London: Baillière, Tindall and Cox. 8¾" x 5½", pp. 480, with illustrations. Price: 27s. 6d.

"Endotracheal Anæsthesia", by Noel A. Gillespie, D.M., B.Ch., M.A. (Oxon.), M.D. (Wis.), D.A. (R.C.S. England); Second Edition; 1948. Wisconsin: The University of Wisconsin Press. 8¼" x 5½", pp. 268, with many illustrations. Price: $4.00.

Diary for the Month.

MAY 25.—South Australian Branch, B.M.A.: Branch Meeting.
MAY 25.—New South Wales Branch, B.M.A.: Ethics Committee.
MAY 26.—Victorian Branch, B.M.A.: Council Meeting.
MAY 27.—New South Wales Branch, B.M.A.: Branch Meeting.
MAY 27.—South Australian Branch, B.M.A.: Branch Meeting.
MAY 28.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute; Brisbane City Council (Medical Officer of Health). Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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